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PROGRESSIVE MUSCULAR ATROPHY.—REPORT OF FIVE CASES IN ONE FAMILY

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The various types of muscular dystrophies have been fully described in the many text books and previous papers and it would be unnecessary repetition to give the classic description of the disease.

The purpose of this paper is to present a family history with five brothers and sisters affected; to present different types in the same family; to stress a particular etiological factor; and to give the results of some endocrine treatment in two of the cases.

FAMILY HISTORY

Mrs. P., age 52, the mother of twelve children, is an apparently healthy woman of middle age, whose family history so far as she knows has been negative for any muscular, mental or nervous abnormalities. She has been in good health all of her life with the exception of excessive nervousness at the menopause which was passed at 45 years.

She began menstruating at 11 years of age. Was married to M. C. at 17 years of age. She gave normal birth to five children by this marriage. Two were premature and died in a few days. Three are living.

Mrs. B., age 34, physically and mentally normal—has four children, the oldest being 14 years of age. All are normal.

D. R. C., age 33, physically and mentally normal, has five children living in good health, oldest is 9 years of age. All are normal. One died suddenly when two weeks of age.

W. C., age 30, physically and mentally normal. Has four children living, all normal. One died at birth, cause not known.

At the age of 25 Mrs. C. married Mr. P. a maternal first cousin. Mr. P. is normal physically and mentally with no abnormal family history. His past life is negative for venereal infections but he has been a fairly heavy user of alcoholic drinks.

During this marriage Mrs. P. has given birth to seven children, four boys and three girls, all living. They are all normal mentally and nervously except case no. 6 who will be discussed in the case history. Of these seven children five have developed some form of muscular dystrophy.

(1) Mrs. W., age 27, normal birth, no childhood illness of consequence except an illness in her third year which began with a bowel condition but later was called "Brain Fever" by her physician. She was ill about 2 months. Her health was good from then till about the twelfth year. The only abnormal condition was bedwetting until 9 years of age. Breasts developed and pubic hair appeared about the twelfth year but menstruation did not begin until the fifteenth year.

During her twelfth year, she developed a pecular gait in walking. This became worse until she could not run and would fall if least overbalanced. She could not raise herself from a sitting or prone position without using hands and walked slowly with a waddling gait. This condition has been slowly progressing to the present time.

At present time she is in fair physical condition. Excepting a chronic nephritis she has no organic abnormalities. Legs are not atrophed but somewhat hypertrophied. She is very weak, especially the thigh, hip and pelvic girdle muscles and has some sway back. Upper arms and shoulders and scapular muscles showing some atrophy and about 50% loss of strength—tendon reflexes absent—Wassermann negative. She is the mother of one child, age 7, which is normal physically at present. This case though beginning in later childhood conforms more closely to the pseudohypertrophic type.

(2) C. P., age 21. Normal birth, no se-

rious illness. Has some curvature of spine since infancy—bedwetting until 10 years of age. Puberty at about 14 years—at ten years of age he began to develop weakness in legs, a waddling gait and fell frequently. This condition has gradually increased. At present time patient is fairly well nourished young man. He walks with difficulty, having a peculiar waddling gait and throwing his feet sideways; also, he has a very decided sway back. The muscles of the legs, thighs, hips and lumbar region are badly atrophied—all deep reflexes are gone—has difficulty raising knees. Muscles of lower legs though somewhat atrophied, still have fair amount of strength. He does not seem to be affected from the waist upward. This case is evidently a pseudohypertrophic type

(3) Miss N. P., age 19—No serious illness—bedwetting up to present time. Breasts and pubic hair developed at 12 years of age. Menses began at 16 years. Had no trouble until 15 years of age when she began to have some weakness in back and a noticeable prominence of scapula.

Physical examination shows only an atrophy of the muscles attached to the lower and medial sides of the scapulae which gives a winged appearance to the scapula. There are no signs of lumbar, hip or leg muscular involvement. This case conforms more closely to the juvenile type of the disease.

(4) C. P., age 16. History negative until seventh year when it was noticed that he would fall easily. Then he developed the typical waddling gait, sway back and difficulty in rising from sitting or reclining position. The process extended rather rapidly in this case and at the age of 13 he was confined to a wheel chair.

Physical examination shows a well nourished boy—practically entirely paralyzed. He can still move his feet and hands but cannot raise knees, move hips or raise upper arms. All deep reflexes are absent. Muscles of the thighs, hips, back, shoulders and upper arms are markedly atrophied. Only the head and neck are not affected. Another pseudohypertrophic type.

This boy was put on adrenal extract three years ago; at that time he was very thin and the disease was rapidly progressing. Since that time the course of the disease has been very slow, sometimes apparently stationary for months and he has gained many pounds in weight.

(5) Miss O. P., 15 years of age—apparently normal at present time but has the same history of genital and breast development at 12 years of age but she has not begun to menstruate yet.

(6) F. P., age 13, has had no serious illness. At 11 years of age he began showing weakness of the hips and thighs. Would fall easily—gradually developed to where he could not run or walk fast. Was put on suprarenal and testis extract last year. The course of the disease has been slowed but is still progressing to a certain extent. He can walk and handle himself well, but shows a beginning atrophy of the thigh and girdle muscles.

He is the only one of the children who seems to be affected the least mentally. He is very perverse and does not keep up in school, having been kept in the same grade at school for the past 3 years.

(7) M. P., 11, normal physically at present time except for especially large calves of legs.

The points I wish to stress are:

1. Five cases in a family of seven children.

2. Blood intermarriage as an etiological factor.

3. Males are affected earlier and the disease progresses faster than in the females.

4. Females develop normally but menses are delayed.

5. Different types in same family—Cases 1 2, 3, 4 and 6 conform more closely to the pseudohypertrophic type while case 3 conforms more closely to the juvenile type.

6. Endocrine products seem to lessen the rapidity of progress and help the nourishment in the two cases in which it has been used.

THE RATIONAL DEVELOPMENT OF MEDICINE

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When one views the high spots of medicine from its very mythological beginning and traces its evolution down the ages up to the present time, he becomes appalled at its slow and rational development. It has been a slow and rather tedious process

for at least two reasons: First, time has been a fundamental prerequisite to the birth of such gigantic subjects; and second, it deals with the most vital spot of the human family, its physical and mental welfare. Its course is too large for proper assimilation, too exquisite for summary by mere human intelligence.

Self preservation and behavior while ill is decidedly an inborn characteristic; as Roswell Park tells us, the lower animals, when sick or wounded, instinctively lessen or alter their diet, seek seclusion and rest, and even in certain cases seek out some particular herb or healing substance. We therefore, realize that the prehistoric animal, less developed, from a physical and mental standpoint, than the highly sensitive human being of today, followed certain helpful procedures akin to modern medicine.

The science and practice of medicine have a dual inception. It remains today as a division between these two; the art of medicine and the science of medicine. Aesculapius, supposedly of Greek ancestry, mythically the son of Appolo and the nymph Coronus, was considered to be the "God of Medicine." Throughout all Greece huge temples were erected to his honor, places where the sick and afflicted could go for relief. Miraculous cures were often reported, and the services of this great person were much in demand. Many of the temples were conducted by Aesculapian priests, they having been endowed with particular powers of healing, and appointed his personal representatives. This perhaps forms to a certain degree the basis of modern psychiatry, because the practice was wholly an art.

Then came the great Hippocrates. "The Father of Medicine." This most remarkable individual was of Greek ancestry, and in his younger days was supposedly a priest of an Aesculapian temple; having had the training associated with the observation of many ill people, and having observed that many of these diseases were consistent in their manifestations, he was the first to place medicine on a rational foundation. He gave to medical literature some of its most classical descriptions of disease. With his efforts began the establishment of "The Hippocratic School of Medicine," which was formed in Athens, and later removed to Alexandria about the year 320 B. C. Such great men as Ptolemy Soter, Herophilus, Heraclides and Celsus probably received their inspirations and guidance from his great work.

Of ancient men, history teaches us that Galen was a most colorful personality; educated among the last of the Alexandrian school, he later moved to Rome. The Roman physician, at this time, was perhaps more skillful than the Greek physician, especially the Roman surgeons. Galen, a Greek by birth, acquired the Roman aspect, consequently being possessor of both Greek and Roman virtues. He was a most fluent and accurate writer, as well as a keen observer. He was perhaps our first great anatomist, and gave to the world many very accurate anatomical descriptions. By force of law, however, his works of dissection were limited to the lower forms of animal life. At this period the dead human being was considered more sacred than the living, and nothing was looked upon with more horror than manipulation of the lifeless human body, thereby interfering with the migration of its soul. He was an outstanding man of his day and is foremost in his time.

Julius Caesar was of great aid in the advancement of medicine. He recognized the value of a healthy and strong army of men. He caused the physicians and surgeons to be elevated socially, by virtue of which their prestige and authority were increased. He attached to his armies medical detachments, and profited greatly by their services.

For the next ten centuries medicine made no progress. In fact there was great deteroriation both in thought and action. Many of the useful discoveries were virtually forgotten, and the art and science of medicine and surgery reverted to ignorance and superstition. Absurd remedies were again used and magic healers proclaimed.

In the twelfth century Emperor Frederick II did a great service to surgery. He deliberately contradicted all past custom and law, issuing a decree that human dissection should be legal. He went further than this and issued an order that at least once in every five years a human body should be dissected in his kingdom. By virtue of this great step many of the mysteries of the human body were clarified and from that beginning a knowledge of the structure of the human body began to be acquired.

The great surgeon Pare was next to make his outstanding contributions which

have immortalized him among men. He treated infected wounds with burning oil, having previously conceived the idea that there was some evil source of life at work among the wounds of man. This treatment was exceedingly painful to the patient, and was followed by a large and frightful mortality. This mortality, however, was not as extensive as that previous to his treatment, because in those days a severe wound was almost certain death. Of particular interest in history, is that event which followed a great battle. Many men were lying wounded on the battle-field. Pare cared for them as quickly and efficiently as he knew how, but long before the last wounded soldier was reached the supply of oil had been extinguished. Knowing nothing else to do, he dressed the wounds of the remaining men with wet dressings and liquid solutions, which must have contained antiseptic solutions. That night his mental condition was so disturbed that he was unable to rest well, consequently he arose early and went to visit his patients with the full expectation of finding those treated in the latter condition to be near death. What he actually found was that those patients treated with burning oils were in great pain and many of them near death, while those treated by his newer method were comfortable and greatly improved. With the institution of such treatment a great contribution was made to the advancement of medicine. Other than this great contribution Pare made a most valuable addition to surgery with the introduction of the ligature for the discontinuation of hemorrhage. As simple as this may seem to us now, we taking it so much for granted, it was a most worthy addition, as the knowledge was not prevalent at that time that blood did circulate in the human body, because this great discovery was soon to follow, and was an evidence of the great work of Harvey.

About this time two great brothers were to make their debut in medicine and leave behind them accurate and definite knowledge which has never been surpassed. These two brothers were William and John Hunter, of England. Although they were great physicians from every angle, they have gone down in history largely because of their outstanding work in anatomy. They had at their disposal an abundance of material, because they so made it. Their original dissections were most exquisite.

Modern medicine, however, probably had its inception with the acquisition of a knowledge of physiology, pathology, bacteriology and chemistry. Pasteur and Koch, remarkable minds; original, creative and conservative, what unusual mentalists they must have been. Their ideas were not born in an instant but developed slowly and with definite progression. And it is with ease that the modern student follows them in their mental process; constant, sure, constructive and reasonable. Their ideas were that organic forces were at war, life is chemistry, disease has a chemical basis with the human family versus toxic forces, manifested through chemistry, liberated from micro-organisms and other sources of energy.

Such discoveries which have given a workable knowledge to the medical profession has certainly made surgery possible. It was for the internist, chemist and bacteriologist to pioneer the way. How helpless the bold surgeon of today would be without the basic knowledge given him by the more particular man of science. He would be helpless in his effort to combat shock, sepsis or any other of the allied conspirators of surgery, without this aid.

How often does one hear such a remark as this, "I hope to see the day when the field of internal medicine equals that of surgery, because in the past few years terrific strides have been made in surgery and so little added in the field of internal medicine." No statement could possibly be more absurd than one such as this. Surgery certainly owes the very existance of its vitals to fundamental medicine. When one observes the course of modern surgery, he cannot help but wonder if it yet continues along those same rational lines upon which it was founded. The science of surgery was first conceived to satisfy a need for emergency, especially during war, to save life. Then as our knowledge was broadened we recognized certain diseases as being surgical. Today, surgery is our one means of saving life in many instances from accidental injury. We have recognized such diseases as acute appendicitis as being surgical, intestinal obstruction and perforated visci. In other words, the application of the science of surgery to the human body, is a process primarily instituted to aid nature in her fight against obstructive and degenerative forces. When it ceases to be this then it is no longer an aid to the natural resources of the body but an added hazard for the

tissues to overcome. The most remarkable characteristic of the human body is its ability of compensation, and when given a chance, in many instances, its response and power of resistance is nothing short of miraculous. One cannot disobey, mechanically, the fundamental laws of physiology and anatomy and arrive at anything but a bitter end.

Frequently one sees cases who have been operated for a so called "fallen colon." or whatever name one chooses to attach to such a questionable condition. The procedure seems to be that of extensive suturing of the colon to lessen its lumen and stay its motility. In a very limited experience the writer has yet to see any but ulterior results from such a procedure. It will, after a while, recede into oblivion just as have the various plastic operations for kidney fixation, and many plastic procedures of the pelvis to say nothing of the epidemic a few years ago of gastric resections.

Recently at the bedside of a patient, a history about as follows was obtained. The patient, a female just past thirty, has had three major operations in as many years. At the first operation the abdomen was opened, the appendix removed and two ovarian cysts removed; about one year later the abdomen was again opened, the round ligaments shortened and a vaginal operation done below; one year later she was again operated, the uterus, both tubes, both ovaries were removed, a "fallen colon" found and extensive suturing apparently done. Today this patient presents a most pathetic picture. Her psychic condition is hopeless, she having been divested completely of the benefit derived from normal function of ovarian origin. There is a partial obstruction of the intestinal tract, its seat seems to be two fold, first at the pyloric end of the stomach, and all along the descending portion of the colon. The procedure of choice here is certainly worse than a puzzle and must have for its solution, one far wiser than I. If further surgery is resorted to, then a gastro-enterostomy must first be done, this will relieve one obstruction, then the whole large bowel must be resected, because there too is a definite obstruction.

At what places the viscera are traumatised, certain damage is done, the tissues are caused to be necrosed, and as a consequence adhesions form. The popularity of the diagnosis, "laparotomy" or "surgical abdomen," is in no small way responsible for the popularization of excessive manipulation. It has offered the surgeon a haven of mental rest and an excuse from persistent effort in establishing a diagnosis, as a consequence the abdomen is opened in the midline with a large incision, the pancreas and intestines investigated, the gall bladder evacuated, the ovaries, tubes and uterus displayed, to say nothing of the veriform appendix, then the procedure is as it should be. This, of course, applies to chronic conditions, and not emergencies. Such cases, as could be more intelligently handled, the seat of dysfunction having studiously been arrived at.

Medicine having arrived at its present and glorious state, over a period exceeding two thousand years, having been born on a basis of conservatism, having been sponsored by an attitude of temperance, clothed in a sheath of tolerance, and undoubtedly directed by the God of nature, should stay free from the bounds of fanaticism. Aesculapian art, coupled with careful management and Hippocratic science, yesterday, today and tomorrow, remains steadfast and placed, and in a vast majority of cases will be rewarded by sound, comfortable and lasting results.

PYELONEPHRITIS IN WOMEN

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ETIOLOGY AND PATHOLOGY

The gynecologist sees pyelonephritis as a complication of pregnancy, and the puerperium and in cases complicated with development of intrapelvic tumors and in post operative cases. It may occur as a sudden development, as an acute attack or very slowly as a chronic condition. This latter condition may follow an acute pyelonephritis, or may arise from a slight but progressive obstruction of a ureter. We observe this at times in women who have a prolapse of uterus with marked cystocele. Usually we may assert there are two causes responsible for the occurrence of pyelonephritis; the first is an obstruction to the outflow of the urine; the second cause is an infection. DeLee claims that bacteriuria is present in 15 per centum of pregnant women. Bacteriuria plus the presence of an enlarging uterus together with the distortion of the ureters incident

to the pregnant state accounts for the comparative frequency of pyelonephritis in pregnant women. It has been estimated that the normal pressure of urine in the kidney pelvis is 10 m. m. mercury and hence slight causes may check or impede the outflow of urine. In about two-thirds of the women coming to autopsy, the ureters, and particularly the right, have been found dilated and filled with urine. The colon bacillus is usually the infecting organism, but the streptococcus, the gonococcus, the tubercle bacillus, the bacillus albus et cetera, have been reported as a cause of pyelonephritis. These organisms are conveyed to the kidney pelvis and ureter in different ways, as by way of the kidney tubules, or excreted through the kidney (bacteriuria), or conveyed through the blood stream, or from surrounding structures (as colon and appendix), or by way of the lymph channels, or by traveling up from the bladder along the mucous membrane, as we previously believed, or even by floating from a lower to a higher level in a quantity of stagnant urine in a ureter whose lumen is obstructed or by way of the peri-ureteral lymphatics to reach the subpelvic areolar tissue which envelops the blood vessels of the kidney at their entrance into the pelvis.

Crabtree and Cabot recently declared that the streptococcus and staphyloccus produce a different set of lesions in the kidney than that produced by the typhoid and the colon bacillus.

The colon-typhoid group causes acute pyelonephritis, acute pyelitis, chronic pyelonephritis, and pyonephrosis, whereas diffuse suppuration, perinephritic abscess, septic infarct, capsular abscess, cortical abscess, and capsulitis are caused by the coccus group. Lesions characteristic of both groups are found in a mixed infection.

Of all the pelvic tumors the rapidly growing tumor of pregnancy is the one most frequently associated with acute pyelonephritis. Occasionally acute pyelonephritis suddenly develops during the course of the growth of a fibroid uterus. Pyeloureteritis may also accompany or follow a post operative cystitis or an acute gonorrhoea or a septic catheterization. It may develop as early as the eighth week in pregnancy. However, it is encountered about the fifth month or later. Sometimes pyelonephritis is seen during the puerperium, and then it may be mistaken for some form of septic infection. More often we find the right kidney involved than the left.

In its manifestation chronic pyelonephritis is less virulent than the acute form of the disease. It arises from a gradually induced stasis of urine accompanied by an infection. The colon bacillus is usually the source of this infection, but less often the gonococcus and tubercle bacillus may be the cause. Again the kidney may be the source of the infection, or it may come from the bladder, from the surrounding parts or by way of the blood stream. The source of the obstruction may arise from a prolapsed uterus and bladder, or from a slowly growing tumor that is compressing or constricting the ureter, or from a calculus in the ureter, or from a contraction or stricture of the ureter as a result of operative trauma.

Symptoms.—Although symptoms like painful and frequent urination often precede the onset, acute pyelonephritis is quite frequently ushered in with chill, pain traversing the course of ureter, and along the back attended with fever. Usually there are present pain on urination, vesical tenesmus, and a diminution of output of urine. Often we have a temperature of 104 to 105 F. and usually a prolonged and exhausting chill precedes this rise of temperature. There is a markedly tender kidney on the affected side, and enlargement may be very perceptible. The presence of large numbers of epithelial cells, pus cells and albumin is found on urinalysis. Sometimes one can palpate the ureter through the vagina as a tender and enlarged cord.

After the lapse of several hours or days there may be quite a reduction of the subjective symptoms, and the temperature declines, the pain subsides, the urine is passed in larger amounts, and it shows an increase of pus and albumin. The condition of the patient may appear to be improving from twenty-four to seventy-two hours when the symptoms may all of a sudden reappear.

At different intervals and with varying intensity the attacks follow one another. Prostration may not be very pronounced in the milder cases, but in the severer ones there is emaciation of the patient, and a hectic appearance in the face. At first usually only the right kidney is involved, but within a few days or a week we may begin to notice pain along the opposite

ureter and evidence of the spreading of the infection in that direction.

The disproportionate increase of albumin as compared with the number of leukocytes in the urine is proof sufficient of kidney involvement. An extension of the inflammatory process to the structure of the kidney is shown by the presence of hyaline and granular casts in the urine.

Cystoscopy reveals an inflamed trigone and congestion or often edema of the ureteral meati. Cystoscopy if performed within the hour of onset may reveal nothing abnormal unless hematuria initiates the disease, when bloody urine may be seen to swirl from the ureters. In suspected cases the ureters should be catheterized and specimens obtained in sterile test tubes for culture. In the acute pyelonephritis of pregnancy and in acute phlebitis or cellulitis following labor or abortion the temperature is intermittent or remittent in type from the beginning and is accompanied by chills.

PROGNOSIS

Acute pyelonephritis may quickly yield to treatment, or on the contrary it may be very stubborn and persistently resist all methods of treatment. Generally the prognosis is favorable and we get recovery in from two to eight weeks. A rapid disappearance of symptoms follows labor and the emptying of the uterus. Pyelonephritis constituting a form of puerperal sepsis may manifest itself during the puerperium. If acute pyelonephritis is due to the presence of pelvic tumors it is cured by the removal of these growths, which relieves the pressure on the ureters. In chronic pyelonephritis the prognosis is determined by the nature of the infection and the organic changes present, e. g., whether the infection is the result of the tubercle bacillus on the one hand or is caused by the colon bacillus on the other; or whether the obstruction can be overcome, or the extent of distension of the kidney pelvis, or the amount of dilitation of the ureter that is present.

In cases of long standing pyelonephritis and in those with recurring or permanent dilitation of the kidney pelvis and residual urine, the prognosis is far less favorable. Local treatment should be employed in such cases, even when no more than temporary improvement is to be expected, when more radical measures are not practicable. Geraghty states that formaldehyde solutions in strengths of 1-5000 to 1-2000 are more effectual than the silver preparations.

TREATMENT

Experience tells us that the treatment of acute pyelonephritis is postural, local, general and medicinal. As to the postural treatment, the patient should be placed in the Sim's position on the well side, and the elevation of the hips should be exaggerated with a hard pillow. The knee-chest position should be required several times during the twenty-four hours. Keep the bowels open. Milk, sweet or butter-milk, and vegetable soups may be taken. She should take water freely. The patient must be kept off of meat broths and solids. The urine should be rendered alkaline in the early stage by administering sodium citrate or sodium acetate 15 to 30 grains every two hours until the urine becomes alkaline to litmus, then every four hours. To obtain alkalization more quickly 15 to 30 grains of natrium bicarbonate is added to the dose of the citrate or acetate. All acute cases are changed to hexamethylenetetramine 10 to 15 grains and acid sodium phosphate every six hours as soon as there is a subsidence of acute symptoms. The reaction of the urine suddenly brought about retards the bacterial growth. Some patients do not tolerate hexamethylenetetramine, but manifest an immediate aggravation of symptoms and the appearance of blood in the urine due to renal irritation and muco-pus from the irritated bladder. The urine is uniformly cleared by a return to alkaline therapy. Some recommend acriflavine (neutral) in enteric coated tablets three times a day. Some patients do well with hexylresorcinol grains 4½ to 9 four times a day after meals and at bed time followed by a glassful of milk. To produce a more continuous and free drainage in obstinate cases the catheter may be left in situ for one or more days. All chronic cases and the acute cases that are not relieved by treatment with medicines in a proper length of time should have the ureter of the affected side catheterized and the ureter and kidney pelvis irrigated with boric acid solution and sterile water, followed by the instillation of 5 c. c. of 1% silver nitrate every one to five days, or 1% mercurochrome, or argyrol 5% to 25% give better results in some cases. Kidd recommends the use of 5 c. c. of 20% neosilvol instilled into kidney pelvis. I can say from experience that this is a remedy one can rely on. Both

medical and lavage treatments are used until three successive kidney specimens are found free from pus and negative to culture. The cases are ambulant except during the acute exacerbation. In unresponsive and chronic cases all foci of infection and predisposing causes must be found out and removed. Calculus in the kidney pelvis or ureter, pelvic tumors and adnexal conditions must be removed to relieve the existing stasis. Kidney ptosis with kinking or torsion must be corrected by suspension. In some cases this latter condition has been and can be relieved by passing a good sized catheter up into the kidney pelvis. Dr. Guy L. Hunner insists upon thorough dilatation of the ureter at the obstructed point if that can be demonstrated. These cases have been successfully treated by continuous drainage with a permanent ureteral catheter for several days or weeks.

If, in spite of all efforts, the symptoms continue and even become more violent and there is almost constant pain, with increasing anemia, and if typhoid-like symptoms develop, one should not hesitate to induce labor. When acute pyelonephritis occurs as a complication of tumors of the pelvis it should be handled in the same manner as acute pyelonephritis of the pregnant state. After there is a subsidence of the acute symptoms, the patient should be operated, so that a repetition of the attacks may be avoided. If after proper rest in bed, and the use of irrigations and urinary antiseptics, the symptoms persist, these pelvic tumors should be operated at once, and nitrous-oxide-oxygen-ether being used for the anesthetic.

The nature and source of the obstruction and the form of infection determine the manner of treatment of chronic pyelonephritis. In tuberculous pyelonephritis it is needless to say that nephrectomy should be done as soon as the diagnosis is made, provided the other kidney is in sound condition. In other forms of chronic pyelonephritis the local treatment should have in mind the relief of urinary stasis in the ureter affected if this exists, the giving of antiseptic solutions, and even the injection of vaccines. The nature of the ureteral obstruction determines the methods employed to relieve the urinary stasis. If external pressure on the ureter causes the stasis, as is seen in pregnancy, postural treatment, as well as ureteral catheterization, may produce the desired results. We must remove tumors and inflammatory masses that press on the ureter. A ureteral stone should first be treated by injecting sterile oil into the ureter, or by dilating with ureteral catheter, or by leaving the catheter, one or more, in situ for one or more days, or if the stone is seen to produce a swelling or tumor in the vesical portion of the ureter it may be removed by fulguration with the diathermy electrode or with scissors.

When the obstruction is due to prolapse of the uterus and bladder we may obtain temporary relief by having the patient wear a pessary, or relief may be secured by operation. When obstruction is caused by infiltration of the broad ligament with carcinoma the condition is usually incurable. When obstruction of the ureter accompanies ptosis of the kidney and kinking of ureter it may be benefited by wearing a belt or pad, or by the suspension operation.

Stricture of the ureter or narrowing of the lumen may be relieved by the use of dilating catheters or by bougies. Indeed, together with antiseptics and diluents, this dilation seems to be all that is needed. In most cases, however, lavage of the kidney pelvis with boric acid solution or silver nitrate solution 1-1000, or colloidal silver 25% will be of great value in producing a cure. The largest ureteral catheter possible should be used when we lavage the kidney. This is used for two reasons: it more completely dilates the narrowed or obstructed ureter and it allows a more complete and efficient washing out of the pelvis of the kidney.

The direct method of cystoscopy and ureteral catheterization in these cases is to be preferred over the indirect method. A little time should be allowed for the kidney pelvis to drain out, after the catheter is inserted. We may then allow the irrigating solution to flow into the catheter by gravity, using a burette or a funnel. As a rule 75 c. c. of the irrigating solution is sufficient to use at one sitting. This treatment may be repeated every three to four days to one to two weeks, according to the effect obtained and the seriousness of the symptoms.

Usually no unfavorable reaction will result if the treatment is given with care and with asepsis. There may be some slight discomfort arise from this treatment, but it will subside in a few hours.

It is well to tell the patient that she *may* have considerable pain in that kidney and ureter for one to three hours, and in some instances the patient may experience very severe pain. But notify her before hand not to be alarmed, but that she may take a glass of hot water every 30 minutes for three times and may use a hot water bottle to the painful area and that there is no cause for alarm. This admonition is sometimes worthwhile. The patient should not be pronounced cured until several urinalyses show the absence of bacteria and pus cells from the urine.

So far as can be ascertained from the literature, the use of vaccines in chronic pyelonephritis has not been encouraging, nevertheless good results unquestionably are obtained in some cases. Vaccine may be employed alone or with other methods of treatment.

A culture should be secured at the time of the first ureteral catheterization, and a vaccine made from this culture, Autogenous vaccines are to be preferred to stock vaccines. However, some claim better results from stock vaccines. From the initial, or immediately following the first ureteral catheterization a portion of this urine should be used to inoculate guinea pigs, in order to rule out tuberculosis.

THE HEART IN SURGERY

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The heart in surgery should be of intense interest to the surgeon. In the mortality list following surgery, heart failure in one form or another is all too frequently given as the cause of death. To my knowledge of such cases, patients so dying have not been given a thorough preliminary examination before operation, hence I cannot see why this diagnosis should have been given.

It is a well established fact that the heart will stand up under surgery even in the presence of severe organic lesions minus block, plus good normal rhythm.

In the series of cases to establish this fact as conducted by Dr. Sprague at the Massachusetts General Hospital many cases even in advanced decompensation were operated successfully; let me quote from his findings: "170 cases were selected for study; there were 42 fatalities, and of this, 22 were heart disease deaths." Understand these were advanced cardiac cases who required surgery. Further findings:

The ability of the patients to carry on the normal affairs of life will tell more about the heart than can be discovered by examination.

No type of heart disease is *per se* a contra indication to necessary surgery.

Medical treatment of a failing heart before operation will in many cases convert a poor surgical risk into a relatively good one. Obese or chronically septic cases are always poor risks.

The behavior of the respiration, pulse, and blood pressure, during and after operation is the best clinical guide to both internist and surgeon.

The skill of the anaesthetist is of the highest importance and greatly influences prognosis, plus the ability of the surgeon to work rapidly and cause as little traumatism as possible.

In light of the above facts why should a normal heart fail as the result of ordinary surgery, or at least why should a heart fail in which there has been no preliminary mention of organic disease placed upon the chart previous to operation, or again a case in which a good prognosis has been made to patient and friends and yet the cause of death given as myocardial failure.

Surely past history or present examination must reveal symptoms of such a condition should such symptoms have been sought for.

Now what is wrong? Is the surgeon negligent in history taking or does he content himself with a cursory cardiac examination. Of course it is understood I am not speaking of emergency surgery, although I know many so-called emergency cases are far from such, and the operation could be postponed while such examination be made as will safeguard the patient and probably influence the prognosis or at least show a patient's cardiac condition.

Let us speak of some of the conditions that place the patient in the danger zone:

Cardiac muscle failure or insufficiency with block or fibrillations. History should give indications of their presence and proper examination with the electrocardiagraph, clinical examination and x-ray

with past history should bring to light the symptoms of myocardial insufficiency.

I cannot go at length into the diagnostic methods, as they are classical and known to you all, but stress the point that such examination should be made and properly recorded on the chart before operation.

In this day and age sclerosis and hypertension are rapidly increasing as evidenced by the ever increasing multitudes of sudden cardiac deaths. Both conditions enhanced by high living and rapid pace of life. And this brings us to blood pressure in surgery.

Moot's rule of blood pressure ratio, should be more widely used. If followed during operation one will get early warning of danger. Many elements have a marked effect on blood pressure according to Ruth & Taylor of Philadelphia, and McKesson, to-wit:

The diastolic pressure alone is of more value than the systolic alone.

The pressure ratio offers the earliest signs of danger.

25 per cent increase in pulse rate, plus 10 to 25 decrease in blood pressure puts the patient in the danger zone.

Many elements have a marked effect on blood pressure ratio during operation.

The emotional and mental condition of the patient.

The position of the patient on the table.

Lack of skilled anaesthetist, length of operation, and oftimes unnecessary traumatism.

McKesson speaking of shock in anaesthesia states that a pulse rate of 100 and rising with progressively falling blood pressure reaching a systolic of 80 mm. or less, if shock continues for thirty minutes or more during operation, without effective remedial measures, death is almost inevitable in twenty-four to seventy-two hours. This guide discloses the onset of shock at least twenty minutes earlier than it is indicated in any other way.

No major operation should ever be performed without the blood pressure machine being attached and readings taken at all times, and more especially is this the case when the age of patient is above forty and the anaesthetist is not an expert. In no other way is it possible to know when the patient passes from anaesthesia to shock.

Blood chemistry should be a routine measure in all patients above forty, at least that is in major surgery.

Let us review the subject of anginal attacks, manifested by acute epigastric pain.

I wonder if any statistics have been made as to the number of such cases operated for gall bladder or chronic appendicitis, I venture their number is legion. A major operation in the presence of such anginal attack is dangerous.

In such cases a painstaking clinical examination plus the electrocardiograph will minimize such mistakes, or at least bring to light the patient's true cardiac condition.

Time is altogether too short for a subject of this magnitude and in conclusion I would make a plea that the patients destined for surgery be given a more thorough cardiac examination and if any doubt exists that more time be taken and that all such findings be charted, for it is too bad when a patient will go to a surgeon for a simple appendectomy or other pelvic surgery, that no record be made on the chart of any past cardiac condition, then see such patient die in the course of three to four days and have such a death recorded as due to cardiac failure.

THE ROLE OF RESPIRATORY INFECTIONS IN DISTURBANCES OF THE HEART

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The part that respiratory infections play in heart disease may yet remain controversial. When one recalls the universal distribution and high incidence of respiratory infections, and then makes a cursory mental survey of cardiac disturbances possibly attributable to such infections, one feels justified in concluding that the occurrence of heart disease either functional or structural during the course of these infections is of little significance to the busy general practitioner. However, bedside observers are familiar with occasional otherwise unexplained cardiac disturbances, occuring during the course of, and following acute respiratory infections. And these cardiovascular accidents

in rare instances will happen in what apparently are otherwise insipid affairs.

For a few years following the 1918 epidemic of influenza, emphasis was placed upon the cardiac phenomena observed during and immediately following the epidemic. There then followed a period of relative quiescence, due no doubt to the absence of an epidemic during this era. The 1928 epidemic was comparatively mild, both as to incidence and severity of the disease. This latter fact probably accounts for the paucity of recorded observations on the late epidemic. Nevertheless, this storm did not pass over without leaving an imprint upon the physical economy of the community.

The present paper purports to recall some of the recorded experiences of observers on the 1918 epidemic, to add a few notes concerning the effects of the 1928 scourge, and to emphasize the importance of careful handling of those respiratory infections occurring in cases of heart disease.

It is not pertinent to the present paper to consider such respiratory conditions as bronchitis and its various manifestations, viz., asthmatic bronchitis, bronchiectasis, pulmonary abscess, tuberculosis, or the pneumonias. It is felt that with the possible exception of the pneumonias, heart disturbance consequent to these conditions is of little import. This paper particularly concerns itself with "influenza" in its various grades of severity.

Since the epidemic of influenza in 1918 there has been a gradual disappearance of old terms such as "la Grippe" and "colds." Sufferers from these former ailments are now included under the term "flu." With this transition there has developed in the minds of both physicians and laymen a distorted conception of the true picture of influenza. Whether or not these milder upper respiratory infections are influenzal will not be considered.

As suggested by Hyman[1] the actual incidence of myocardial affection resulting from influenza is quite difficult to ascertain. Quoting statistics from Berlin, London, New York and Boston, he finds that the incidence varies between 4 and 7 per cent. Were it possible to get accurate figures, the incidence would no doubt be considerably higher than these figures would indicate.

Anatomically, any part of the heart may be affected by influenza. Influenzal valvulitis, although rare, has been accurately described by Oppenheimer[2]. There are no doubt other cases on record.

The myocardium may be, and frequently is, the site of predilection for the toxin. The marked and persistent convalescent asthenia with low systolic blood pressure, enlarged heart, tender liver, and cyanosis, in cases of influenza is evidence of a damaged myocardium, even though more classical symptoms or signs of myocardial failure may be absent. Structural changes in the myocardium as a rule are not demonstrable even in fatal cases. However, in the absence of hypertrophy, an enlarged cardiac area which is quite common is accounted for at postmorten by dilatation. Dilatation means a stretching of the muscle fibers beyond physiological limits, resulting in loss of tone and circulatory failure. This probably accounts for many fatalities.

Primary affection of the pericardium is not seen at the autopsy table. But the absence of acute pericardial lesions demonstrated at autopsy does not necessarily exclude involvement of the pericardium in sublethal cases. In this connection it is interesting to note that Musser and Herrmann[3] in a review of 1720 consecutive necropsies performed at Charity Hospital in New Orleans, in a five year period from 1921 to 1925 found an incidence of 17.7% pericardial adhesions. It is worthy of note that this study was made on deaths occurring in the years immediately following the epidemic of 1918. Locke's[4] figures for a similar study in Boston reported in 1916 were 12.6%. The high incidence of pericardial adhesions recorded at the autopsy table, and in many instances in cases who during life were not suspected of having such lesions, suggests strongly a possibility of undiagnosed pericardial disease in cases of influenza. Adhesions between the pericardium and pleura when so placed as to prevent freedom of mobility of the heart may result sooner or later in myocardial embarrassment.

The toxin of influenza not infrequently affects the conduction system. The effect is primarily that of depression rather than excitation. In the milder cases referred to as "simple" postinfluenzal bradycardia by Hyman[5] the slow heart is likened to that which follows typhoid fever. As pointed out by Hyman, this is probably a result of changes in the sinus node rather

than a result of involvement of the extrinsic nerves of the heart, because other evidences of symphathetic imbalance in these cases are not demonstratable. Depending upon the severity of the toxin various grades of conduction disturbances may be found. There is no relationship between generalized toxic effects and conduction defects,—that is to say, a severe grade of arrhythmia being quite possible in an otherwise mild infection. In the more severe affections varying degrees of sinoauricular block, auriculoventricular block, and even complete dissociation of auricular and ventricular rhythm may be encountered. During the late 1928 epidemic of influenza I had occasioin to observe two or three cases of bradycardia during the height of the disease when the patient was febrile. Unfortunately, electrocardiographic studies were not available. From a review of Hyman's record[6] it would seem that as a rule the severer grades of conduction disturbances are encountered in those individuals past middle life, and it would be expected that the irregularity would be more likely to persist in this group than in younger individuals.

Still another cardiac manifestation of influenza is angina pectoris although scarcely mentioned in the literature. The incidence of this syndrome following influenza is sufficient to warrant close study. Two per cent of the cases of influenza studied by the Committee of the Witkin Foundation[7] developed anginal symptoms. It has been pointed out that the age incidence of postinfluenzal angina is lower than from other causes. Hyman's records show that quite frequently angina occurs in those individuals who have previously had no symptoms of heart disease. The severity of the angina is indicated by a mortality in his small series of 33 1-3%.

The question naturally arises as to the possible outcome of these influenzal conditions. A resurvey of eight cases of postinfluenzal heart disease made by Hamburger and Priest[8] would indicate that the majority have a favorable outlook. Of the eight cases which were rechecked after three years, five had completely recovered, two were not traceable, and one was unimproved. While this is a small group, they confirm the observations of White, Romberg and others referred to by Hamburger. From a comparison of the reports by Hamburger et al, and Hyman one might summarize in a general way with the statement that the prognosis depends upon three factors, namely: (1) Age, (2) the anatomic and functional integrity of the myocardium upon which the infection has been superimposed. (3) the amount of permanent structural damage done by the toxin in a given case.

The following cases will serve to illustrate some of the points brought out in the above discussion, and to emphasize the importance of the occurrence of respiratory infections in the cardiac cripple:

CASE REPORTS

Case No. 52, a male, farmer, age 25, presented himself for examination on August 24, 1929, with a complaint of "pain in the heart." First symptoms began nine years ago following an attack of "flu" of one week's duration. At that time he developed a pain in the region of the heart which lasted for six or eight months. This gradually disappeared. He was told at the time that he had a "murmur." In January, 1929, there was a recurrence of the pain, which in every respect simulated the former pain. The only other complaint was epistaxis. He is of menial habits and smokes eight to ten cigarettes per day, and denies the use of stimulants. The family history is negative for circulatory diseases.

Physical Examination revealed a normal temperature, a pulse rate of 100, blood pressure of 140/76 in a sthenic individual with a panniculus subcutis measuring ½ to 1½ cm. Reflexes were hyperactive. The tonsils were cryptic. The apex beat was neither visible nor palpable. Sinus arrhythmia with an occasional extrasystole was noted. The heart sounds were somewhat distant and there was a sticky first sound at the apex. The carotid reflex was markedly positive. Shifting of cardiac dullness with position could not be demonstrated.

X-Ray Examination of the chest revealed an obliteration of the left costophrenic angle with an apparent fixation of the apex. There was distinct left sided heart enlargement.

The Electrocardiogram showed the presence of right ventricular ectopics, prominent P2, a depression of S-T2, and negative T3.

In spite of the fact that fixation of the electrical axis in this case could not be demonstrated, the case was diagnosed in-

fluenzal adhesive pericardio-pleuritis with consequent cardiac hypertrophy.

Comment: The occurence of a "murmur" with the former attack is worthy of note. Quite often in the course of a physical examination the physician observes rather definite alteration in one or other of the cardiac sounds that cannot be classified as true murmurs. These are usually recorded as murmurs, but may in many instances be external manifestations of pericardial changes. It is now well known that pericardial adhesions, when existing over a period of time, can cause cardiac hypertrophy. The left sided heart enlargement in this case may be accounted for on such a basis.

Case No. 89, is that of a male, age 52, whose former occupation was clerical, but now dairying. He presented himself for examinaton on December 24, 1929, with a complaint of irregular heart. The cardiac history dates back to 1916 when, while undergoing an insurance examination, he was told that his heart was slightly irregular. There were no symptoms at this time. He was examined again in 1922 when he was told that the heart was fluttering. The circulatory symptoms began about January, 1929, when, following a mild attack of "flu," he noticed a thumping in the left side of the chest synchronous with the heart beat. Shortly after this he was told by his physician that he had a bad heart. In March, 1929, he began to notice that he fatigued easily. He is now annoyed by an oppressive feeling over the heart after eating a heavy meal. Dizziness is also complained of. Other symptoms are denied. The habits are regular.

Past History: Pertussis and chickenpox during childhood; polyarthritis in 1921 which was relieved by the extraction of several teeth; a neurofibroma was removed from the arm in 1921. He suffered a generalized run-down feeling with loss of weight five years ago which was relieved by a tonsillectomy. He denies venereal diseases. He has been married twenty-one years and there are no children. The wife had two miscarriages soon after marriage. The patient states that a pelvic condition in the wife prevented their having children.

Family History: The father died at 65 of heart trouble; the mother died at 72 of liver trouble with jaundice. The remainder of the family history is irrelevant.

Physical Examination: The physical examination revealed a tall hyposthenic individual with a panniculus measuring from ½ to ¾ cm. and of firm musculature. Blood pressure was 150-130/80 and the pulse rate was 61 and totally irregular. There was moderate arteriosclerosis of the fundal arteries and the peripheral arteries. The auscultatory rate of the heart was 100 with a pulse deficit of 39. The apical sounds were ringing and valvular in quality. The aortic second sound was accentuated. A systolic apical murmur transmitted toward the aortic region was elicited. There were no thrills.

Electrocardiographic Studies revealed an auricular fibrillation with a rate of 120 and frequent ventricular ectopics. The T. waves were varible as to direction and amplitude in all leads.

Comment: This case is worthy of note. From his story it is evident that he has had an arrhythmia for several years. However, compensation had apparently been well maintained until an otherwise insignificant mild respiratory infection occurred which reduced the cardiac reserve to the extent that a beginning failure resulted.

Another case is worthy of note in order that too much emphasis cannot be placed on the effect of so-called "flu" on an already damaged myocardium.

Case No. 20, male, age 60, was admitted to the hospital on April 14, 1929, complaining of shortness of breath and weakness. His story is that he had a mild attack of "flu" in December, 1928. He states that he has never felt quite right since that time. However, he has been able to carry on his daily activities until ten days prior to admission when he began to notice a progressive shortness of breath on exertion, that was accompanied by a generalized body weakness, some slight restlessness, and a slight pitting edema of the pretibial regions. It was further noticed that there was considerable gas on the stomach and the patient would attribute all the symptoms to this latter discomfort. Anorexia has been present since the onset of the present illness. He denies the occurrence of heart pains. There has been some blurring of vision since the attack of "flu." A slight cough is also complained of, beginning at the time of the present illness. Diminished frequency and quantity of urine has been noticed during the past few days. Bilateral frontal morning headache has been pres-

ent for several years. He denies the use of alcoholics, stimulants, or drugs.

Physical Examination revealed a man of apparent age 65, who was suffering from marked air hunger. There was a pasty pallor of the skin, and cyanosis of the lips, fingertips and the toes. A slight pitting edema was noticed in the pretibial regions. Examination of the fundi oculi revealed a marked sclerosis of the fundal arteries and a slight blurring of the discs. The lungs showed signs of edema at both bases anteriorly and posteriorly. The heart extended to the nipple line on the left side in the sixth interspace and to the parasternal line in the fourth interspace on the right side. There was increased manubrial dullness. The peripheral arteries were markedly thickened. The heart sounds were over active and there was marked accentuation with a metallic note of the aortic second sound. The heart rate was 120; the blood pressure 234/130. The peripheral arteries were thickened and tortuous, and the pulse assumed the character of a Corrigan. The liver was felt about two fingers breadth below the costal margin. The right lobe of the prostate was somewhat enlarged and hard.

The red blood cells numbered 3,860,000; hemoglobin 72%; the blood N. P. N. was 50.81 mm per 100 cc of blood. Specific gravity of the urine varied between 1.010 and 1.017; albumin was present on all of three examinations; hyaline and granular casts were found.

Diagnosis: Arteriosclerotic myocardial disease with hypertension and congestive failure no. 3; vascular nephritis.

Progress: The patient remained in the hospital about two weeks. The blood pressure remained at a high level, varying from 232/126 to 200/114. He left the hospital somewhat improved as evidenced by a clearing of paroxysmal attacks of dyspnea and a decided diminution in the edema.

Comment: A patient with known cardiovascular disease who develops an upper respiratory infection, however mild, should be kept abed much longer than those who are not the victims of such disease. There is no means of estimating the extent of damage done to the myocardium, and the severity of the attack of influenza is not a criterion of myocardial damage. It is well therefore to insist upon a prolonged period of physical inactivity in that patient who is known to have had cardiovascular disease prior to the attack of "flu."

Heart disease may exist for many years and not cause the patient any distress. This is true of certain of the valvular lesions, more particularly the mitral valves. The mitral valves may be damaged during childhood in the course of an attack of inflammatory rheumatism, and an anatomical alteration will be demonstrable at any subsequent examination of the patient throughout life. These patients, however, will carry on their daily activities without any distress so long as the myocardium remains intact. While it is quite true that the continued strain of valvular insufficiency gradually breaks down the myocardial reserve, the actual break is frequently initiated by an otherwise harmless mild upper respiratory infection that the patient will term an attack of "flu."

The following case serves to illustrate this point.

Case No. 87, male, age 40, had an attack of acute inflammatory rheumatism at the age of 13, followed by milder attacks at 14 and 15. At the end of his college career he was told by a physician that he had heart disease. Since that time he has lived in various parts of this country, at high and low altitudes, and in South America following his profession of geologist, and he has suffered no particular distress. He frankly denies any symptoms that would suggest congestive or anginal failure prior to the present illness. He would date the present illness back to December, 1928, when he had a mild attack of "flu." This attack was of little significance to the patient and the services of a physician were not obtained. Since the attack of "flu" he has complained of a feeling of lassitude and gastric distress. He would term his general feelings as below par. During the second week in April, 1929, he noticed periods of skipped heart beats, and he states that he has been conscious of the heart beats, noticed more particularly after retiring and when lying on the right side. During this period he has also had momentary knife-like pains in the precardial region; slight shortness of breath on exertion is also complained of. He has been accustomed in the past to playing 18 holes of golf in an afternoon. Diagnosis at the time of the examination was rheumatic mitral insufficiency with congestive failure No. 1 and mild anginal failure. An electrocardiogram taken at

the time was not remarkable except for an occasional supraventricular ectopic complex and a negative T3. The patient has been under fairly constant observation since his first visit and the general progress has been downward. In November, six months from the time of the first examination and eleven months from the time of the attack of "flu," he developed auricular fibrillation which was diagnosed electrocardiographically. Since that time a considerable part of the time has been spent in bed and there has been a gradual narrowing of the physical sphere of this patient.

Comment: This patient has reached the age limit for this type of lesion. Nevertheless, since he has been extremely cooperative with all physicians who have attended him at various times, even though he did not have a physician during his mild attack of "flu," a word of advice in the matter of prolonged rest in this type of condition might have been a means of prolonging life. In such cases every effort should be exerted toward the maintenance of integrity of the myocardium.

Case No. 57, a housewife, age 34, was first seen July 30, 1929, complaining of "heart pain." She states that since an attack of "flu" in December, 1928, she has never felt the same. In addition to such general symptoms as lassitude, fatiguability, gastric discomfort, she has had two phases relative to the circulatory system. The first phase immediately followed the "flu," extended over a period of three months, and was marked by several attacks of syncope, shortness of breath on exertion, a fullness in the precordial region, numbness in the left arm, and transient slight edema. of the lower extremities. Following this period there have been attacks of heart pain occurring at irregular intervals, associated with exertion and excitement, and marked by a severe cutting pain in the apical region of the heart which seems to radiate to the posterior chest on the left side. This pain is of momentary duration, but is followed by numbness in the left arm which lasts for several hours. During one of these attacks her family physician administered amyl nitrite which aggravated the symptoms. She was told by her physician that there was an irregularity of the pulse during one of the attacks.

She further complains of a constant tight feeling in the precordium that is aggravated by exercise and excitement. She sleeps on three pillows and cannot lie on the left side. The past history includes an operation for goiter, growing pains during childhood, typhoid fever at 15, and "uremic poisoning" prior to delivery of a normal infant eleven years ago.

Physical Examination revealed a hyposthenic individual 5 feet in height, weighing 137 pounds, with a pulse rate of 84; blood pressure of 120/86. The heart on percussion extended 10 cm to the left and 4 cm to the right. Supracardiac dullness measured 8 cm. The heart was rhythmical, the sounds were distant, the first being inaudible at the base. Aortic second sound was accentuated.

X-Ray examination showed a slight widening of the aortic shadow.

Electrocardiogram was normal.

A diagnosis of postinfluenzal angina with possible early coronary sclerosis was made. The diagnosis was strengthened when the patinet's symptoms were relieved following the administration of theophyllin-ethylenediamine.

Comment: The occurence of angina in an individual of this age is worthy of note. The past history suggests previous damage of the heart. However, the angina together with other symptoms of myocardial weakness, is new and was apparently initiated by the attack of influenza.

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THE TREATMENT OF CELIAC DISEASE FROM THE STANDPOINT OF VITAMIN DEFICIENCY*

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The description of Celiac disease is just the same now as that given by Gee in 1888. The literature of today does not enlighten us as to the true cause any more

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than early writers on the subject. Gee compared it with endemic diarrhea alba of India.

Celiac disease may be cunfused with two or three other conditions presenting abdominal enlargement; that is, if you have never seen a case. Quite often the child starts vomiting after being fed—projectile in character, suggestive of pyloric spasm or stenosis. As these conditions develop in the first few weeks of life, and Celiac disease in late infancy and early childhood, pyloric spasm and stenosis can be excluded very easily. Hirschsprung's disease may be confused with Celiac disease, but this condition is congenital and the child has suffered with constipation from birth: the colon is dilated and the abdominal distention is enormous. Tubercular peritonitis is associated with severe abdominal pain. The abdominal enlargement is gradual; there is wasting, pallor and weakness.

The Celiac child is one who has been a difficult feeder. on the bottle. The condition never develops with a breast-fed child; if so, it is several months after the child has been weaned. The majority of cases occur at fifteen months, or as they approach their second year. It may occur as early as six months and as late as from three to five years. The symptoms and appearance of the Celiac child are quite characteristic, and with a careful history and examination, one is not liable to confuse it with any other condition of early childhood presenting abdominal distention.

The child is brought to the doctor, due to the character of the stools, which are loose—not formed, not watery, but bulky, pale in color—looking very much like oatmeal porridge or gruel, losing weight, protruding abdomen. These are the clinical symptoms when we first see the child.

In reviewing the present-day literature on the subject, the symptomatology, regardless of text or writer, is about the same. Some may go a little more in detail in the laboratory findings; but, on the whole, the clinical description and findings are the same as those given by Gee in 1888.

Regardless of the etiology, there seems to be an inability of the Celiac child to digest fat, sugar and starch, and for this reason, some of the investigators have attributed the cause to a deficiency of bile entering the intestine, pancreatic deficiency, overfeeding with fat, sugar and starch, functional disturbance of the nervous supply of the abdominal viscera, causing a disturbance of digestion and the absorption of toxic products.

Sauer states that the disease has never been produced experimentally, and cites a case of a child developing the condition on visiting a cousin who had the disease, which leads to the hypothesis that it might be transmitted from one child to another. This I believe to be quite impossible, as there is no other case on record. Perhaps these children's environment and diet being the same, it might be considered as a coincidence.

Post-mortem evidence has been of little assistance in placing the cause except to demonstrate the absence of any organic disease. To me, Celiac disease may be classified as one of the nutritional disturbances brought about by deficiency of the vitamins. This is shown by the retardation of growth and development. While there is no true rickets present, there is delayed formation of the centers of ossification of the epiphyses of the bones. The teeth are subject to early caries, the muscles are atrophic and weak and the child is very irritable, showing a nervous manifestation. There is a marked degree of anemia. While the disease is active, the child ceases to grow, is frail, stunted; these are symptoms of vitamin deficiency and to me, are more plausible for the cause of Celiac disease, than any yet given.

Vitamins are chemical substances of vital importance for the maintenance of the health of the body and are derived from the vegetable kingdom. Clinical experience shows that many pale, tired-looking children with unhealthy skins, whose appetites have been bad, improved rapidly after placing them on diets containing sufficient vitamins. R. McCarrison, London, in his experimental work, has proven quite conclusively that vitamins constitute one of the few essential agents for keeping the body alive and by their lack in proper balance, there is loss of appetite, indigestion, diarrhea alternating with constipation, inflammation of the colon, loss of weight, general weakness and lack of vigor, headache, tendency to edema and unhealthy skin, subnormal temperature and cardio-vascular depression.

The retardation of growth may be explained by the inability of the child to di-

gest whole sweet milk, which supplies the fat soluble Vitamin A. The diet lacking this can produce a wide range of physical disorders—children are undersized, retarded in development, more susceptible to bacterial invasions leading to bronchitis, pneumonia and even tuberculosis. Cod liver oil furnishes Vitamin A in a large amount, but the majortiy of these children cannot take cod liver oil due to its causing a digestive disturbance. Vitamin A is found most abundantly in that which the child cannot tolerate—butter and cream—and to me this is the outstanding cause of retardation of growth and arrested development in Celiac disease.

With a deficiency of Vitamin B, there is anorexia, lowered vitality, fretfulness, restlessness, pallor, low hemoglobin, and growth retardation. Presence of Vitamin B improves the appetite and enables the child to assimilate food to a greater advantage.

Water-soluble Vitamin C is contained in fresh fruit, green vegetables and tomatoes. A deficiency of this vitamin results in the development of scurvy. Still of London reports several of his cases having had scurvy preceding the symptoms and development of Celiac disease, which precludes the probability of Celiac disease being a condition of vitamin deficiency.

Vitamin D, the antiricketic vitamin is found in milk, butter, eggs, animal fat, vegetables, cod liver oil and irradiated ergosterol, which is perhaps the richest source of this vitamin. Vitamin D represents a force and power in the building-up process of the body, and delayed growth of bone indicates what has been proved a doubt—the connection of this vitamin with calcium and phosphorus metabolism and the prevention and cure of rickets.

In the treatment of Celiac disease, the textbook states that "We are dealing with a chronic affection and there is no tendency to a spontaneous recovery. Cow's milk is not only unsuited; it is the least suited. If the patient can be cured, it must be by diet; high proteins, low carbohydrates and fats."

Miller of London states that "Fresh cow's milk, cream, butter, meat, fat, must be omitted for many months. If exacerbation occurs they should be ommitted for years. The course is always slow, whatever may be its end."

Sandor A. Levinsohn says, "Treatment unsatisfactory; no method is always good; try and experiment. Medication is of little value."

Sidney V. Hass divides the treatment into two parts: Keeping the gastro-intesinal tract free from toxic accumulation by castor oil weekly, and a colonic irrigation daily with sodium bicarbonate. Second, dietetic, plain milk should *not* be given. Protein milk, lactic acid milk, dry milk prepared with a fat-free milk may be given. Carbohydrates should be avoided, also fat. Protein may be used in all forms and apparently any quanity. Bananas may be used in any number—as high as sixteen a day.

Sauer states, "Up until 1923, results in treating Celiac disease were unsatisfactory. At that time, the late John Howland instituted protein milk. A tablespoonful of protein milk per pound body weight is given in twenty-four hours, gradually increasing until the child received the number of calories he should have for a normal child of the same age. Ringer's solution is used to add minerals. The mixture may be sweetened with vanilla or any other flavoring to encourage its palatability.

Julius Hess divides the treatment into three stages: First, protein milk alone; second, protein milk reinforced by almost pure protein food; third, that in which carbohydrates are added, is really a difficult one.

Kerley, of New York, in 1924 reported fifteen cases of Celiac disease to the American Pediatric Society, and in the April, 1930, "Archives of Pediatrics," six additional cases, treated by a mixture of water, 30 oz., casein, 2¼ oz., starch, 2¼ oz., sugar, 2¼ oz. This proved more satisfactory in his hands than protein milk in liquid or dry form. He states that he has no trouble in handling this diet and before these cases came under his care they had been tried on various milk preparations, including protein milk, and they were in an advanced condition of malnutrition. He states that if the abdomen still be enlarged after many months, this may be corrected by the application of Basler's abdominal support.

To carry out my theory that Celiac disease is a condition due to viamin deficiency, it will be necessary for me to report a case, going into minute detail of the diet, where *all* the vitamins were early added to the treatment, including sweet

milk in evaporated form; carbohydrates in the form of Vitavose; whole lactic acid milk, with the object of adding fat; saur kraut juice to furnish acid in the form of lactic and acetic; minerals—calcium and phosporus, and vitamins. Also, Spintrate, a powered spinach, one teaspoonful of which is equal to a well-rounded tablespoonful of the average cooked spinach.

Spintrate contains an excellent source of iron, calcium and phosphorus. It is a very rich source of Vitamin A, B and G. Information given by the manufacturers of Spintrate states that it is close to dried yeast in its content of Vitamin B and G, and by their tests, they find it about twice as rich as dried brewer's yeast in antineuritic value, and about equal to it in antipellagric value.

REPORT OF CASE

L. M., female child, 17 months. Family history, father and mother living, healthy, one sister, eight years old, healthy; normal birth, breast fed for four weeks, then modified cow's milk, and at six months put on undiluted cow's milk, unboiled, never was able to take cod liver oil, never cared for vegetables, but was very fond of mashed potatoes, and these she ate in large quanities. The mother said she was advised that the child could have all the potatoes she wanted, and at times would make a whole meal on potatoes alone.

When the child was fifteen months old, she weighed twenty-six pounds, and at this time started vomiting, losing weight, bowels somewhat loose, and the abdomen distended. The mother says she never had any fever, stomach becoming more enlarged, the only way to reduce it, by enemas. She had a great deal of gas, which the mother says she could hear, rolling. The appetite had been good, the child slept well and woke about once during the night.

Physical Examination: The child was very cross and irritable, greatly underweight and loss of turgor; ribs and scapula quite prominent; unable to walk, weight, 18 lbs., 3 oz., weight of a normal child eight months old, and she should have weighed 22 lbs., 12 oz. The forehead was very prominent and the hip circumference small. The abdomen measured 20 inches, chest 18½ inches, temperature, 99½ per rectum. Blood findings, hemoglobin, 44; w. b. c., 10,200; r. b. c., 3,200,000; urine, negative.

At seventeen months, the disease was then of two months' duration. The child had been on lactic acid milk; Hess' modification of milk (Carnation milk, lemon juice, yolk of egg) was on this for about ten days and then changed to Kerley's treatment. which consists of casein, 2¼ oz., starch, 2¼ oz., sugar, 2¼ oz., and water, 30 oz. This mixture the child could not handle, and was continuing to lose weight.

I started on the first day's treatment, with: Water, 30 oz., lactic acid milk, 6 tbs., protein milk, 4 tbs., saccharine, 1 gr., to the mixture; total calories, 390. Of course, on this number of calories, I did not expect the child to gain, but was testing her out to see how she would respond to the treatment that I expected to institute.

On the following day, I added 2 tbs. of protein milk and 2 oz. saur kraut juice.

Three days later, I added protein milk, 8 tbs., lactic acid milk, 10 tbs., and kraut juice, 2 oz. and 6 bananas.

Four days later, the child was taking 15 tbs. protein milk and lactic acid milk, 8 tbs. with 1 tbs. Vitavose.

Eight days later, she was receiving 19 tbs, protein milk, 6 tbs. lactic acid milk, 2 tbs. Vitavose. At this time, Spintrate was included, and 3 oz. of kraut juice. The child at this time weighed 21 pounds, 6 ounces, a gain of 3 pounds, 3 ounces in eight days.

On the ninth day, skimmed milk was added, one pint, to make up the quart of fluid.

On the eleventh day, one quart of boiled cool skimmed milk replaced the water in the solution and protein milk, 22 tbs., lactic acid milk, 6 tbs., kraut juice, 4 oz., Spintrate, 6 tbs., and Vitavose, 3 tbs.

On the nineteenth day, the child's weight was 22 pounds, 4 ounces. Vegetables were added by cooking with boilable protein milk and skimmed milk; the bottle feeding was not changed. The diet remained the same and during the first three weeks, scraped beef, baked chicken, hard-boiled egg, bananas, and orange juice were added together with the milk mixture and the total calories amounted to 2235.

At about the sixth week, evaporated milk was added to the milk mixture in the following manner: Skimmed milk, 1 qt., evaporated milk, 12 oz., kraut juice, 5 oz.,

protein milk, 30 tbs., lactic acid milk, 15 tbs., and Vitavose, 10 tbs., Spintrate, 5 tsp. The diet has remained about the same, with exception of the addition of liver, baked sweet potato and shredded wheat biscuit.

During the period of treatment, the child has received no enemas, castor oil or any medication. At this time she is two years of age and has been under my supervision for eight months. Her height is 35½ inches, weight 29 pounds; normal height, 33 inches, weight 26 pounds. Weight for the four winter months (December to March) remained stationary due perhaps to her inability to take sun baths.

On this diet, the child has never had any remission of the symptoms, and has gained in every respect. Whole lactic acid milk was early added to the diet, with the thought that the protein milk would help to overcome action of the fat in the acid milk and the lactic acid neutralizes the buffer substance in cow's milk, making it more easily digested. Evaporated milk was also included early, as it occurred to me that the fat in this milk, due to its very fine globules, would be assimilated and digested as easily as mother's milk and in acid media, with lactic acid milk and kraut juice, would probably promote digestibility and at the same time would be adding Vitamins A, B and D. These vitamins are not destroyed in the preparation of evaporated milk. As much as 24 oz. of the milk have been added to the 24-hour feeding mixture without any disturbance, as is reported experienced by authorities on the subject. Up to this time, fat was added very cautiously and it was said months or years were required before these cases could assimilate fat satisfactorily.

Sugar, one of the important food elements which the Celiac child has been unable to digest and had been advised to add very cautiously, was introduced into the diet in the form of Vitavose, with more than satisfactory results. It is a wheat-germ sugar and very rich in Vitamin B, containing the other mineral elements and soluble-nitrogenous compounds present in the wheat germ. As large an amount as 12 tbs. were added to the 24-hour mixture without any ill effect.

After the child had been on the diet outlined for eight months, I omitted powdered protein milk and lactic acid milk from the formula. I continued with evaporated milk, Vitavase, Spintrate and kraut juice, to note the reaction, if any, and my instructions to the child's mother was for equal parts of evaporated milk and water in the formula.

A week later, the child was brought to the office and upon verifying the formula with the mother, I found that she had misunderstood me as to the amount of evaporated milk she was to use and that week had prepared the formula with two parts evaporated milk to one part water. The child had taken the mixture without any ill effect or remission of the condition on the addition of fat, as predicted in the textbooks. Inasmuch as the child relished and digested the mixture satisfactorily, I allowed her to remain on the 2:1 milk and water proportion (plus, of course, Vitavose, Spintrate and kraut juice) in addition to which she receives a meat, fruit and vegetable diet.

The large proportion of evaporated milk erroneously added to the milk formula determines that the fat in evaporated milk must be as easily digested and assimilated as mother's milk.

This case of Celiac disease improved more rapidly than any I have seen or found reported in literature; this I attribute to treating and feeding a child with this condition, as a vitamin deficiency.

CONCLUSION

1. Celiac disease is, in all probability, a vitamin deficiency.

2. Fat, in the form of evaporated milk, can be added early to a high protein mixture with an acid media.

3. Sugar can be added early in the form of grain-germ sugar.

4. Iron, calcium and phosphorus, also vitamins A, B and G may be added to the food mixture in the form of Spintrate.

5. Kraut juice may be used as an acidifier, also adding additional minerals and vitamins.

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PERFORATED GASTRIC AND DUODENAL ULCER

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In all the field of surgery there is probably no condition more dramatic in onset, more fulminating in its course or, if untreated, more tragic in outcome, than perforated gastric and duodenal ulcers.

The subject of peptic ulcer is one of intriguing interest and there is a constant changing of ideas and opinions by a great many of our most exhaustive workers in this field—on the part of the internist as well as the surgeon. This is particularly evidenced by the fact that Deaver, who, up until two years ago, routinely did gastro-jejunostomy following closure of the perforation, now practices only simple closure of the perforated duodenal ulcer unless the closure itself produces obstruction or stenosis of the lumen.

There can no longer be any doubt that all non-perforated ulcers should receive at least a trial medical treatment because it is a well known and well recognized fact that many peptic ulcers properly treated and properly managed will heal and remain healed.

We frequently see patients who have had definite signs and symptoms of peptic ulcer for years and these are the cases that are so difficult to manage and one of the most difficult points to determine is—when does it cease to be a medical problem and when does it become a definite surgical entity? This is a question which probably will never be satisfactorily decided.

However, there is one type of ulcer in which there can be no doubt as to the necessity for immediate surgery. I refer to the perforated peptic ulcer which is always a definite surgical condition and deserving, as well as demanding, immediate and effective surgical relief if one is to succeed in averting a most certain fatality.

Moynihan classifies or describes perforations as acute, subacute and chronic. The acute type gives way suddenly and completely and usually presents a fairly large opening which allows the stomach and duodenal contents to escape easily and rapidly into the peritoneal cavity.

The subacute type is described as perforating almost as suddenly but may give prodromal symptoms of exaggerated pain for several days before perforation and the opening is usually somewhat smaller and may be instantly closed by a plug of omentum or in a few hours by fibrinous exudate allowing very little soiling of the abdomen by the gastro-duodenal contents. It is a well known fact that this type of perforation may sometimes permit recovery of the patient without resorting to surgery but this result is one that can never be foretold and the only cases diagnosed as perforated ulcers which should not receive surgical relief are those who refuse to give their permission for operation.

The chronic type is one which slowly eats its way through the coats and allows a protective peritonitis to develop and prevents the spilling of the gastric contents into the general peritoneal cavity. Perigastric abscess may form and since the chronic perforations are frequently on the posterior surface, the resulting abscess is usually subphrenic. It is fortunate, indeed, that the majority of the acute and subacute perforations occur on the anterior surfaces where they are usually easily accessible. It occasionally happens that the posterior perforations are so inaccessible that effective closure is physically impossible.

The Diagnosis of Perforated Ulcers is not usually of extreme difficulty as the great majority of them present a fairly consistent picture and, if one is careful to elicit an accurate history, approximately 90% give a history of previous indigestion or so called stomach trouble varying anywhere from a few weeks to ten or fifteen years. However, there are a few who claim they have never had any previous trouble. Henry P. Brown, in report-

ing a series of 100 perforations at the Pennsylvania and Presbyterian hospitals of Philadelphia, says that ten claimed they were free from all symptoms prior to perforation. The onset is extremely sudden and has been described as a sudden severe pain like the thrust of a dagger in the epigastrium.

This excruciating pain is located usually about half way between the Xyphoid process and umbilicus and from one to two centimeters to the right of the midline. There is occasionally a referred pain to the chest or shoulders, but most frequently to the left supra-clavicular fossa, but, when present, is usually so transitory that the patient makes no particular note of it. The pain is followed almost immediately by an involuntary rigidity of the abdominal muscles which, for the first hour, is more marked in the upper portion of the right rectus muscle but rapidly becomes generalized and is commonly referred to as board-like in its character and the patient resents the approach of the examining hand for fear of increasing the intensity of the pain.

The Breathing is short, shallow and costal in type and the patient keeps his legs flexed and the body is maintained in a rigidly fixed position, as any movement or manipulation causes increased pain and the patient may remain for hours without moving to any appreciable extent. Nausea and vomiting are present in most cases.

The Pulse Rate is an interesting feature in this condition and in many causes the loss of several valuable hours. Immediately following the rupture and for several hours the pulse rate may remain normal or even below normal due to stimulation of the vague nerve. The expression is terror struck and there is extreme prostration, in contrast to what is usually described as shock which is not present for several hours, as evidenced by the low pulse rate, normal blood pressure and normal temperature. For the first six hours the white cell count is of little value in the diagnosis but later shows a rapidly increasing leukocytosis.

Obliteration of Liver Dullness is a symptom which is occasionally present due to a layer of air over the liver. This is sometimes demonstrated by the flouroscope, and, when present, is of considerable value in aiding to verify the diagosis. Shifting dullness due to the presence of fluid in the abdominal cavity and rapidly increasing dullness in the right flank are additional symptoms of value in making an accurate diagnosis, especially in duodenal perforations.

While it is always a matter of great personal satisfaction and pride for our pre and post-operative diagnoses to check, it must be remembered that a human life is at stake and that too much time must not be lost in the effort to be absolutely correct in our diagnosis. It is quite sufficient to be able to decide that the lesion in question presents an acute surgical emergency.

The early symptoms are followed in a few hours by the beginnig of peritonitis. By the second or third day there is true shock and the abdomen is distended and tympanitic, there is cyanosis, the pulse is thread-like and rapid, the extremities are cold and it is quite obvious that the usual result of non-operated cases is inevitable.

These symptoms, in addition to those of chronic ulcer, are usually sufficent to justify a diagnosis of perforated peptic ulcer. Briefly, these additional symptoms may be tabulated as follows: indigestion, epigastric pain or distress, occurring from two to four hours after meals and often about four or five o'clock in the morning and relieved by food and alkalies; seasonal recurrences of symptoms usually in the spring and fall and lasting from four to six weeks with comparative freedom from symptoms until the next attack, loss of weight, vomiting of blood or blood in stools, in addition to the x-ray findings and the analysis of the gastric contents in those cases which have previously been investigated. While it is quite true that in about 80% of perforated ulcers the diagnosis is comparatively easy, there are a number of lesions which may be easily confused with perforations. In Brown's series of 100 cases, acute appendicitis was diagnosed seven times, acute cholecystitis five times, intestinal obstruction in three, and acute pancreatitis in one. In 1901 Moynihan reported a series of 49 cases in which 18 were diagnosed as acute appendicitis.

These were all duodenal perforations and there is usually earlier involvement of the right side than in gastric perforations. This is due to the well defined course the escaping fluid takes as it invades the peritoneal cavity. It escapes onto the upper surface of the transverse Mesocolon to the right of the promontory

formed by the fitting of the transverse colon to the greater curvature of the stomach. This, quite naturally, causes the fluid to run to the right of the hepatic flexure and from there down the right side of the ascending colon to the right iliac fossa, where it may cause the exquisite pain and tenderness so characteristic of acute appendicitis. The fluid then fills the pelvic cavity, next the left iliac fossa and by gradual extension, the entire peritoneal cavity.

Other conditions which occasionally cause some difficulty in differentiation are ruptured ectopic pregnancy, cholelithiasis, acute cholecystitis, torsion of ovarian tumor, gastric crises of Tabes, and occasionally, the onset of the menstrual period. Ruptured ectopic pregnancy may simulate perforated ulcer but may usually be ruled out by obtaining a history of pregnancy and examination may reveal an enlarged uterus, soft cervix, and uterine bleeding. The pain is usually in the right or left lower quadrant and it should also be remembered that perforated ulcer is rare in women, the percentage being about 20%. There may be little or no rigidity.

Twisted ovarian tumor usually causes low abdominal pain and may be accompanied by very little rigidity. Bi-manual examination may reveal the presence of the tumor.

Intestinal obstruction as a rule does not cause a great deal of difficulty in differentiation except in the extremely early stage. The onset is not so sudden and the immediate prostration is not nearly so marked as in perforation. The nausea and vomiting are more marked, which with visible and audible hyperperistalsis followed later by fecal vomiting with distention of the abdomen are quite sufficient to elimnate a diagnosis of perforation.

In acute pancreatitis the pain and prostration are more marked than in perforated ulcer and may present bluish patches of cyanosis over the abdomen and limbs, a symptom which occurs only in acute pancreatitis. The rigidity is less marked and limited to the upper abdomen and the tenderness is more marked on the left side than on the right side. The pulse is usually quite rapid and thready from the onset.

The cholelithiasis attack is in marked contrast to perforation in which the body is rigidly maintained in one position, while the victim of gall stone colic tosses about in an effort to obtain some relief by changing his position. The tenderness is located further to the right and the pain frequently radiates around the ribs to the right scapula, to the right shoulder or straight through to the back. There may be a history of previous jaundice.

Acute appendicitis differs from perforation in that the previous history is not suggestive of ulcer, the rigidity is not so intense and it usually occurs first over the right lower quadrant and may not develop for several hours, whereas, the rigidity of perforation is almost simultaneous with the occurence of the perforation. The pain is not so excruciating, the respiration is not of the quick, jerky, shallow, costal type, and the relative tenderness is quite different in intensity and location.

It is quite obvious that *the treatment* is prompt and effective surgical relief consisting of some form of closure of the perforation which will produce the least amount of narrowing of the lumen of the duodenum or stomach. There is no stereotyped or standard procedure or technique which is effective in all cases and according to one's judgment, the technique may be modified according to the requirements of the case.

There are several factors of genuine importance to be considered which may influence one's choice of procedure. The most important is the length of time elapsing from the time of the perforation until the operation as the mortality rate increases in direct ratio to the number of hours which have elapsed until the patient is operated. Other factors are the location, size, character and amount of induration around the ulcer, the virulence of the escaping fluid, the surgical facilities, and the judgment, experience and skill of the operating team. Since the majority of these perforations are duodenal and since most of them are easily accessible, a simple closure is sufficient. If the ulcer is of the large, thick, battle scarred, callous type, then it is obvious that a gastro-enterostomy or some type of short-circuiting operation is necessary.

The majority of patients with perforated ulcers are in a condition of extremis and since the operation is essentially a life saving measure, it is quite natural to assume that the simplest and quickest method which will effectively close the perfora-

tion is the method of choice. In approximately 85% of cases this can be done by simply inserting a purse-string suture and invaginating the edges of the opening and then planting an omental graft over the repaired area. In most cases this simple procedure is quite sufficient. While the simple purse-string suture, especially in the soft type of ulcer, is one of the most effective means of rapid closure, there are a number of other methods which are equally as effective. Some men use a single interrupted suture going through all the coats of intestine or stomach, while others use a single mattress or several interrupted sutures covered by an omental graft. In duodenal perforation where there is considerable edema and infiltration and the sutures do not hold well, Moynihan describes a method of closure in which the gastro-hepatic omentum is drawn over the anterior surface of the duodenum and fixed at its lower border by a few sutures and the extreme right portion of the great omentum is then turned upward over the duodenum and fixed to form a second protective layer over the gastro-hepatic omentum. In difficult cases this may be followed by closing the pylorus and doing gastro-enterostomy.

There are cases in which closure of the ulcer produces a definite obstruction or stenosis of the duodenum and in these cases it is an absolute necessity to do some type of short-circuiting operation, which always adds considerable time to the operation, and, therefore, to some extent may increase the mortality. The chief controversy involves the question of doing routine gastro-enterostomy following closure of the perforation and there are many arguments for and against this procedure.

To quote from Olson and Cable, some of the arguments against gastro-enterostomy are:

1. It is unnecessary as the perforation nearly always cures the ulcer.
2. It adds to the mortality.
3. There is danger of spreading the infection.
4. It is not necessary in all cases.
5. Reperforation, hemorrhage and stenosis are very rare.
6. The danger of gastro-jejunal ulcer.
7. Brenner favors simple closure in the soft ulcer type and gastro-enterostomy in the caloused type.

And in favor of gastro-enterostomy are the following:

1. Perforation, with closure alone, does not cure the ulcer in a large number of cases.
2. In properly chosen cases gastro-enterostomy does not affect the mortality.
3. The danger of spreading infection is theoretical rather than practical.
4. Closure of perforation always narrows the lumen and gastro-enterostomy safeguards against secondary perforation and the effect of subsequent stenosis.
5. The occurence of gastro-jejunal ulcer is extremely low.
6. Gastro-enterostomy relieves tension on the suture.
7. Co-existing ulcers are favorably influenced.

Immediately after the operation the patient may be given a retention enema consisting of about ten ounces of saline, which may be repeated every four hours or saline may be given continuously by the Murphy drip method. No fluids should be given by mouth for twelve hours or preferably twenty-four hours, unless gastro-enterostomy has been done in which case fluids may be given within four hours. Hypodermoclysis of saline may be given if needed and if the patient is in shock 200 cc of 10% glucose solution intravenously is especially effective. After the patient reacts from the anaesthetic he may be elevated to the Fowler position to drain the fluids away from the diaphram. After twenty-four hours he may have more water or if desired a little tea and after forty-eight hours he may have fruit juices and the diet may be increased as rapidly as the patient is able to tolerate it. It must be remembered that the diet and medical care are most important from a post-operative standpoint and upon this care depends to a great extent the ultimate success of the operation and freedom from recurrence of symptoms.

The question of drainage is one of considerable interest and some controversy.

Moynihan rarely ever drains his cases if operated upon during the first twelve hours after perforation, while Deaver advocates drainage in practically all cases. It has been conclusively proved that the escaping fluid is sterile for the first six hours, and, if there is very little spilling of the fluid, it is rarely ever necessary to drain. The only case which I have closed without drainage developed a rather fulminating peritonitis, a paralytic ileus and the wound drained most freely for some several weeks which was eventually followed by recovery, but I am naturally very hesitant to close without drainage after such an experience.

In all cases the fliud should be removed either by suction or by hot moist saline sponges. Particular attention should be given to the upper abdomen beneath the diaphram to avoid, if possible, the danger of subphrenic abscess or of a septic inflammation spreading through the diaphram and causing an acute pleurisy or empyema.

Drainage, when used, should extend into the pelvis through a suprapubic stab wound. This may also be supplemented, if it seems necessary, by stab wound drains in the right and left iliac fossa and also through the incision.

I would like to very briefly report four fairly typical cases of perforated peptic ulcers, three of which were duodenal and one pyloric:

Mr. H. H., a white man, age 55, occupation farmer, was brought to the hospital, a distance of about 15 miles, in an ambulance. His chief complaint at time of admittance was severe pain in the right side of the abdomen, more marked in the right lower quadrant, but was preceded by a sudden severe epigastric pain followed by prostration. He gave a history of stomach trouble for the past five years which was worse in the spring. The pain came on when the stomach was empty and the taking of food was accompanied by relief of pain until two or three hours after meals. He had never taken any alkalies or other medicine nor had he ever been under any treatment. W.B.C. were 16,800; polys 85; S.L. 11; L.L. 4; urinalysis negative: blood pressure 125/70; temperature 99.8. Immediate operation was advised and the abdomen was opened twelve hours after the onset of the acute pain. There was a moderate amount of free fluid and gastric contents in the free peritoneal cavity and examination quickly revealed a duodenal perforation on the anterior surface about two centimeters below the pyloric ring. It was easily accessible and was closed with a purse-string suture and by fixing an omental graft over the perforated area. All fluid was removed and the abdomen was closed with no drainage. The patient developed a virulent peritonitis and the wound drained pus freely for about three weeks, a paralytic ileus occurred about the fifth post-operative day and ten days after the operation he developed pneumonia. The patient eventually recovered and was dismissed sixty-three days after operation.

Mr. W. E. D., a white man, age 34, occupation farmer, was admitted to the hospital complaining of epigastric pain, nervousness and general weakness. He gave a history of stomach trouble which first occurred 18 years ago, but had no trouble for the past three years. The pain occurred suddenly after lifting a heavy weight. On examination the temperature was 99 .2; pulse 80, and respiration 20. The abdomen was quite rigid and very tender on pressure over the epigastrium. The blood count revealed 15,000 W.B.C., with 81 polys, 17 small and two large lymphocytes. The urinalysis was negative. Operation revealed free fluid in the abdomen and there was a perforation about 6 millimeters in diameter on the anterior surface of the duodenum about 2 cm. below the pyloric ring. This was easily closed and and reinforced with omentum and the abdomen drained. The patient made an uneventful recovery and left the hospital in good condition 19 days after the operation.

Mr. J. S., a white man, age 35, occupation freight handler, entered the hospital complaining of severe epigastric pain which was referred to the right side with a history of previous indigestion and upper abdominal pain occurring several hours after meals and relieved by food and alkalies. About three days before admission to hospital, his pain had been worse. Examination revealed rigid abdomen, tenderness on pressure, especially in epigastrium and over appendiceal region. W. B. C. were 15,600 with 79 polys, 16 small and 5 large lymphocytes. Urine showed trace of albumin and otherwise was negative. Operation was advised immediately and examination of the duode-

num in this case also revealed a perforation just below the pyloric ring on the anterior surface. Purse-string suture sufficed to close the opening and omental graft was fixed to duodenal wall over the perforation. The patient was dismissed in good condition in 13 days.

Mr. C. D., a white man, age 37, occupation plumber, was admitted to the hospital complaining of severe pain in epigastrium which came on suddenly with a history of previous trouble for ten years. The abdomen was typical in its rigidity, the respiration was short, shallow and costal in type, and the pain was quite severe. The blood count was 13,700 W.B.C., 78 polys, 19 small and 3 large lymphocytes. Operation revealed an anterior perforation about 1 centimeter proximal to the pyloric valve. The opening was easily closed with a purse-string suture and omental graft applied in the usual manner. This was followed by a very smooth and uneventful recovery and the patient was able to leave the hospital eleven days after operation.

With the exception of the last, which was quite recent, these cases have been seen at fairly regular intervals and all report that they have been relatively free from previous symptoms. However, they have been warned in regard to their diet and one case reports that it is necessary for him to take a little alkali at intervals but quite rarely as compared to the amount taken before perforation.

In closing I would like to say that this paper makes no claim for any original work and presents more or less of a resume of the experiences and opinions and judgment of various workers in the field. No tables of statistics have been presented due to the necessity of brevity. Suffice it to say that we should make a strenuous effort to make an early diagnosis and insist on immediate operation for the mortality increases in direct ratio to the number of hours elapsing from the time of perforation until operation. Almost all cases operated within six hours recover while about 75% operated as much as forty-eight hours after perforation fail to recover.

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Finally Dextri-Maltose is never advertised to the public but only to the physician, to be prescribed by him according to the individual requirements of each baby.

CALCIFICATION OF VESSELS IN DIABETES

Roentgenographic evidence of calcification of the vessels was present in the legs in 53 per cent of 324 diabetic patients varying in age from 2 to 81 years examined by L. B. Morrison and I. K. Bogan, Boston (Journal A. M. A., April 27, 1929). In the third decade 6 per cent of the patients showed vascular calcification, and in the seventh decade 87 per cent. Twenty-one per cent showed advanced calcification. Advanced calcification was not found under 40 years of age, although definite calcification was present in five cases. No case of gangrene was found under 40 years of age. Syphilis, dental infections and arthritis appear to play no part in the production of vascular calcification in this series. Seventeen per cent of patients without calcification (average age, 41 years) and 49 per cent of patients with calcification (average age 59 years) have blood pressures over 150. Patients over 50 with sclerosis are about twice as apt to have high blood pressure as those without calcification. Six, or 9 per cent of the patients with diabetes of ten years' duration, did not show calcification. Morrison and Bogan conclude from these observations that the incidence of vascular calcification increases with age and with the duration of the disease, and is higher in diabetic than in nondiabetic patients. The degree of the calcification increases as a rule with age and duration. The blood pressure in this series of diabetic patients increased with age but not with duration. Roentgen examination is an accurate method of judging the presence of calcium in the vessel walls. It is more reliable than clinical methods in the diagnosis of vascular calcification. Rentgenograms of the extremities would be helpful to insurance physicians. Diabetes mellitus is an etiologic factor in the production of vascular calcification.

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Articles sent this Journal for publication and all those read at the annual meetings of the State Association are the sole property of this Journal. The Journal relies on each individual contributor's strict adherence to this well-known rule of medical journalism. In the event an article sent this Journal for publication is published before appearance in the Journal, the manuscript will be returned to the writer.

Failure to receive The Journal should call for immediate notification of the editor, Barnes Building, Muskogee, Oklahoma.

Local news of possible interest to the medical profession, notes on removals, changes in address, births, deaths and weddings will be gratefully received.

Advertising of articles, drugs or compounds unapproved by the Council on Pharmacy of the A. M. A., will not be accepted.

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EDITORIAL

THE NEW SCHOOL CHILD

Perhaps the matter applies to all school children, rather than those just entering school, but it certainly is of interest to everyone to protect, at least, the interest of the child just entering school, who in a few weeks will be thrown in contact with many and diverse infections and conditions.

The Journal has always intended it to be a part of its civic duty at this particular time of year to remind physicians, especially the family physician, who has the confidence of the parents and of the children, that much may be done to protect the child in many ways. It goes without saying that every child can be and should be, largely if not wholly, protected from certain infections by the intelligent use of the various prophylatics and preventative medicines. Toxin anti-toxin, occupies a successful niche in the prevention of diphtheria, and the Schick test is so simple in the hands of the qualified that failure in its use is hardly excusable. Small pox vaccine is so well known to both physician and laymen that it would not seem necessary to mention that phase of the matter, yet there are no doubt thousands of school children, today, not vaccinated.

Theoretically, the low water level in Oklahoma as well as in many other states, should be followed by high concentration of solids and with that a high concentration of any infectious matter which the waters may contain. Hundreds of country schools in the State have very poor water supplies, as a rule simply bored wells, subject to any and all types of infections. Health officers and physicians have a large influence in combating these dangers; anti-typhoid vaccine is so simple, so harmless that its lack of use is also inexcusable.

Hundreds of the new children entering schools will be found to have various dental and nasopharyngeal infections and defects, probably the leader of them all is the infected tonsil, accompanied by the usual adenoids, after which it is a race as to whether dental or other throat and ear conditions cause the most trouble. Very good physicians are prone to overlook infections of the ear which may tend to illness of any degree of severity and sometimes end in fatalities.

It should be the pleasurue and the duty of every physician as a professional man and as a citizen to point out in his most discriminating and effective manner the great worth and absolute need of the various boons of preventative medicine to his clientele.

"ROGUES' GALLERY"*

According to reports it required more than six months to catalogue the "Rogues' Gallery." The New York City department of health index includes the names, aliases and records of over 25,000 medical quacks, "cure all" manufacturers, nostrum peddlers, diet inventers and all others who

prey on the ill and guillible well. This massive work was compiled by the National Better Business Bureau at the request of the Department of Health.

It is said that the thousands of folders disclose the life history and careers of abortionists, medical quacks, charlatans; manufacturers of questionable medicine, worthless hair growers, and other tonics; peddlers of "Indian herb" concoctions; harmful cosmetics; including rouges, powders, creams, washes, toilet waters, lipsticks, etc.; manufacturers and peddlers of "horse collars" and other useless electric devices; "inventors" and peddlers of worthless diets, exercise and reducing systems; and a complete list of the manufacturers and analysis of tonics, patent medicines and other medical articles offered for sale in drugstores and other shops, which do not have the approval of the American Medical Association.

It is said that the efforts of Commissioner Shirley W. Wynne to rid the radio broadcasting field of medical quacks and other charlatans really marked the beginning of the work, for at that time he advocated the compilation of an "Indexus Expurgatorius" to be used as a guide by owners of broadcasting stations.

All of this is well and commendable but only applies to New York City. However, it is significant and commendable that the Middle West, through the efforts of the Kansas City Star, the American Medical Association, the Kansas State Board of Medical Examiners, and others are waging a bitter fight to exclude from broadcasting some of the most notorious charlatans and quacks ever allowed existance under the broadfolds of our Constitution. It is more than irritating to note that despite the arrogant quackery and cruel frauds imposed by the Kansas Broadcasters, the efforts to dislodge them are met on every hand by various obstructions, legal objection and injunction.

It is certainly the duty of the physician, for no one else is able to do so, to put such imposters out of business; but the months, and sometimes years, pile up in the attempt to perform such services, rendering the work disheartening indeed.

*The New England Journal of Medicine. Volume 203, page 286. Aug. 7, 1930.

Editorial Notes—Personal and General

DR. HUGH JETER, Oklahoma City, announces the removal of his offices to 218 Osler Building.

DR. W. S. IVY, Duncan, has returned from Denver, Colorado, where he took a special course in medicine and surgery.

DR. ALFRED R. SUGG, Ada, announces the removal of his office to 106 East 13th Street. Practice limited to Urology and Gynecology.

DR. JACKSON BROSHEARS, Lawton, is reported seriously ill, suffering from a respiratory disease resulting from a relapse of summer influenza.

DR. D. H. O'DONOGHUE, Oklahoma City, announces the opening of his office for practice limited to Orthopedic Surgery and Fractures, 912 Merical Arts Building.

JEFFERSON COUNTY MEDICAL SOCIETY met July 7, guests of the Waurika physicians. After diinner the following program was given: Uterine Atresia and Report of a Case, Dr. J. I. Hollingsworth; Rational Treatment of Abortion, Dr. W. M. Browning; Use of Digitalis and Strophanhus in Heart Affection, Dr. C. M. Maupin. October 1st will be the date of the next meeting.

CORRECTION—"Chemical Hysterectomy." In our last issue attention was called to the work of "Smith of New Orleans" on the above matter. This was an error, the pioneer New Orleans authority in the application of zinc chloride for the eradication of certain types of malignancy and other growths of the cervix and uterus was Dr. Ernest S. Lewis, Emeritus Professor of Obstetrics and Gynecology, Tulane University School of Medicine.

AMERICAN SOCIETY FOR STUDY OF GOITER

At the recent meeting of the American Association for the Study of Goiter at Seattle, Washington, Doctor William F. Rienhoff, Jr., of Johns Hopkins University, Baltimore, Maryland, received the annual award of $300 for the best essay dealing with the goiter problem. Doctors O. P. Kimball, of Cleveland, Ohio, and E. P. and D. R. McCullagh, Cleveland Clinic Foundation, Cleveland, Ohio, and Robert P. Ball, of the University of Louisville, received honorable mention.

CANCER CLINIC AT BALTIMORE, MD.

To My Colleagues, the Editors of the Medical Journals of the United States and Canada:

There will be a meeting in the ballroom of Belvedere Hotel in Baltimore, Maryland, Monday, Tuesday and Wednesday, September 15, 16, and 17, 1930, beginning Monday morning at ten o'clock and ending Wednesday evening at nine o'clock, daylight saving time. During these days there will be lantern-silde demonstrations, with four lanterns and screens, on the Diagnosis and Treatment of Diseases and Tumors of Bone.

The first day will be devoted to the fundamental and essential knowledge of the benign and malignant lesions of bone, such as osteitis fibrosa, giant-cell tumors, osteomyelitis, sarcoma and so forth. On the second day, the subject will be the different diseases of single bones, such as the lower end of the radius, vertebrae, etc. The third day will be reserved for the presentation of rare lesions of bone difficult to diagnose. Any member of the medical profession attending this meeting may register such a case by addressing Miss Maude Walker, Secretary to Dr. Bloodgood, Surgical Pathological Laboratory, Johns Hopkins Hospital, Baltimore, Md., enclosing the X-ray films or lantern slides of them (if possible the latter) and sections of tissue, if any. Any member of the medical profession interested in the diagnosis and treatment of lesions of bone is invited.

On account of the size of the ballroom the number must be limited to 800.

Those who wish to attend should write the Belvedere Hotel and register, either requesting the usual rates for a single or double room with and without bath, or the special rates for three or more in a room with and without bath, and the special restaurant rates for a club breakfast, luncheon and dinner. You are advised to bring the answer received from the Manager of the Belvedere Hotel with you and present it when you register. For any further details in regard to this demonstration, address your letter to Miss Maude Walker, named above.

I am very anxious that this invitation should reach radiologists, surgeons, pathologists, and internists who are interested in the subject but have only rare opportunities to observe lesions of bone. In three sessions of two or two and one-half hours each, on three days, with four lanterns and a very remarkable and educational motion picture, the subject can be presented in an almost unforgetable way, emphasizing the essentials and fundamentals in the diagnosis and treatment of bone lesions. All cases registered for presentation on Wednesday, will be sent later to Dr. Bowman C. Cromwell, Director of Clinical Research of the American College of Surgeons, who is Chairman of the Bone Sarcoma Committee. You should become familiar with this registration of sarcoma of bone, if you are not, because you can register all your cases there and receive the diagnosis of a committee, and you can send for groups of bone tumor cases which have been registered, for personal study.

It is impossible except in the largest clinics, for any radiologist, pathologist, surgeon, or internist, to become familiar with the changing clinical, X-ray and microscopic pictures of diseases and tumors of bone as they come under observation earlier and earlier after the first injury or first symptom, and to learn how to diagnose and treat them in the best way.

Sincerely yours,

JOSEPH C. BLOODGOOD.

DOCTOR J. C. SMITH

Dr. J. C. Smith, pioneer Catoosa physician and Tulsan, died July 20, 1930, after several months' illness. He was 54 years old.

Dr. Smith came to Catoosa from Pennsylvania in 1901, and had been active in civic affairs there until he was forced to retire from active practice because of ill health, last year. He was widely known in Masonic circles in Rogers and Tulsa counties and served as county commissioner of Rogers County.

Funeral services were held at the home, Tulsa, with the Rev. C. W. Kerr, pastor of the First Presbyterian church officiating. The body was taken to Neosho, Mo., for burial. Special rites were conducted by the Masonic lodge of Catoosa.

Surviving besides the widow, is one brother, and sister.

RESOLUTION ON THE DEATH OF DR. J. C. SMITH

WHEREAS, in His infinite wisdom, the Great Physician has again permitted that great foe of physical man, Death, to enter our ranks and remove from our midst a brother physician, Dr. J. C. Smith of Catoosa, Oklahoma; and,

WHEREAS, by his long association with us in the practice of medicine and surgery in Rogers County, by his membership in our medical society, Dr. Smith had greatly endeared himself to us,

THEREFORE, BE IT RESOLVED, that we, the members of the Rogers County Medical Society, deeply regret his untimely passing while in the prime of life; and,

BE IT FURTHER RESOLVED that a copy of these resolutions be spread upon the minutes of the society, a copy furnished to the State Medical Journal, and a copy be furnished the family of our deceased brother.

Dr. R. C. Meloy, Chairman.
Dr. W. F. Hays.
Dr. F. A. Anderson.

Rogers County Medical Society,
W. A. Howard, Secy.

DOCTOR ERNEST H. LAIN

Dr. E. H. Lain, Lindsay, pioneer physician of Garvin County, died July 15, 1930, in Oklahoma City, following an operation for cholecystitis.

Dr. Lain was born at Wingo, Ky., August 6, 1878. His preliminary education was obtained at Wingo High School. He graduated from the University of Louisville in

April, 1905. State Certificate issued in July, 1908.

TO THE GARVIN COUNTY MEDICAL SOCIETY:

Whereas it has pleased the Supreme Physician to call from his earthly labors our beloved friend and co-laborer, Dr. E. H. Lain, of Lindsay, Oklahoma, who was a member and constant attendent upon our organization's conventions for many years, yielding the profession of which he was an honored member his unstinted loyalty and untiring effort, and;

Whereas, his wife and child have lost a kind and loving husband and father, and Garvin County an outstanding member of the Medical profession, and the profession itself has suffered an irreparable loss in the death of Dr. Lain:

Therefore, be it resolved that the Garvin County Medical Society hereby tenders to the bereaved family our deep and abiding sympathy in this their hour of great bereavement, and express it as the sense of this body that in the loss of Dr. Lain the medical profession of this County has suffered a distinct and irreparable loss, and the community in which he lived a loyal, upright and honorable citizen, as well as an eminent physician and surgeon:

Be it further resolved that a copy of this resolution be sent to the family of the said Dr. E. H. Lain, at Lindsay, Oklahoma, and that a copy be furnished the local newspapers and the Journal of the Oklahoma Medical Association, of which the said Dr. Lain was an honored and beloved member for many years past.

Respectfully submitted,

Dr. N. H. Lindsay.
Dr. W. P. Greening.
Dr. G. L. Johnson.

The above resolution was unanimously adopted by the Garvin County Medical Society on this the 16th day of July, 1930.

John R. Callaway, Secretary.

TUBERCULOSIS

Edited By
L. J. Moorman, M.D. and Floyd Moorman, M.D.
912 Medical Arts Bldg., Oklahoma City

Bronchiectasis. Alton Ochsner, M. D., Amer. Jr. Med. Sciences, March, 1930.

It is the author's opinion that bronchiectasis is the most frequently encountered chronic pulmonary affection, occurring even more often than pulmonary tuberculosis.

The etiology of bronchiectasis is varied. Cirrhosis of the lung, acute infectious diseases, such as influenza, pertussis and measles, chronic sinusitis, stenosis of the bronchi and chronic bronchitis are all causative factors.

The most characteristic pathologic finding in bronchiectasis is dilatation, of which there are three types: cylindrical, fusiform, and saccular. The lower lobes are the most frequently involved, probably because of the stasis of the retained secretion.

Symptoms and signs as described in most textbooks undoubtedly represent a late stage of the condition. A patient who has had an antecedent pulmonary infection expectorates large amounts of fetid sputum—this is associated with fever, anorexia and loss of weight. The early symptoms and signs are relatively insignificant. Hemoptysis is a frequent symptom, being present in from 50 to 70 per cent of cases. In the majority of cases and in all early cases, the physical findings are minimal. Almost invariably, however, there is some lagging on the affected side, especially at the base. There is no change in the percussion note except in late cases. Diaphragmatic excursion is usually normal. Auscultation may reveal moist rales at the base of the lung. In the advanced cases marked limitation of motion is present; there is impairment of resonance over the affected lobe; moist bubbling rales are audible; bronchial breathing is present.

The introduuction of lipiodol by Sicard and Forestier, in 1922, as a contrast substance has facilitated early diagnosis of bronchiectasis. It made possible the visualization of the tracheobronchial tree by means of the Roentgenray thus permitting the demonstration of any deviation from the normal outline of this system. At the present time it is generally accepted that a diagnosis of bronchiectasis should not be made unless positive roentgenographic evidence is obtained following the introduction of a contrast substance in the tracheobronchial tree.

The treatment of bronchiectasis has been both medical or conservative and surgical or radical. Bronchotomy, because of the almost uniformly bad results, has been almost entirely abandoned. At present the most widely practised surgical procedure is some form of surgical collapse. Artificial pneumothorax, it is generally conceded, should not be considered a curative measure so much as a preliminary procedure, which if not successful, may be followed by more radical surgery.

Bronchiectasis limited to a single lobe is ideally treated by extirpation of the involved lobe. Because of the frequent bilateral involvement and the high mortality resulting from such a procedure, relatively few cases can be treated.

The medical therapy in bronchiectasis has been unsatisfactory; however, postural drainage is of distinct benefit. The repeated intratracheal introduction of iodized oil is of therapeutic value. One hundred and twelve cases of bronchiectasis were treated. Thirty-two per cent were symptomatically cured; 12 per cent showed radiographic evidence of cure; 36 per cent obtained symptomatic relief but following an acute respiratory infection had a temporary relapse; 32 per cent were improved, and are still under treatment. The "passive" method of introducing the iodized oil is the method of choice because of its simplicity and ease of performance. It consists of anesthetizing the anterior pillars of the pharynx, which abolishes the swallowing reflex. Because of the abolition of the swallowing reflex, swallowing is impossible, and the larynx, which normally rises during deglutition to lie beneath the epiglottis

and the base of the tongue, remains immovable. The iodized oil taken into the mouth is then aspirated into the trachea and bronchi.

Primary Carcinoma of the Lung. James Alexander Miller and Oswald R. Jones. Amer. Review of Tuberculosis. Dec, 1929.

Pulmonary cancer was almost unknown until the 19th century, when it was recognized with the development of studies in pathological anatomy. Stokes was the first to use percussion and auscultation for the direct demonstration of pulmonary cancer during life. According to him, frequent and severe bronchitis, resisting the usual methods of treatment, is suspicious of malignant tumor. He considered the best diagnostic sign to be flatness and immobility of the lung without auscultatory evidence of ulceration.

Most authors agree that carinoma of the lung is much more frequent in males than in females.

The origin of pulmonary cancer has been attributed to an overgrowth of a foetal bronchiectasis, atelectatic pulmonic tissue, influenza, pneumonia, old tuberculous lesions, and dust of the street, but these various theories have very little basis of proved fact.

Primary carinoma of the lung may be divided into three main groups: (1) Carcinoma of the lining epithelium of the bronchi, (2) Carcinoma of the mucous glands of the bronchi, (3) Carcinoma arising from the pulmonary alveoli. Grossly, it may by its growth infiltrate simply the lung, or it may also constrict the bronchus, causing secondary pathological changes in the lungs, such as atelectasis, bronchiectasis, necrosis and pleural thickening. It may be associated with inflammatory changes such as an exudative pneumonia, or an interstitial pneumonitis, and a secondary pleural effusion, either serous, bloody or purulent is frequent.

Widespread metastases to other parts of the body occur from a primary pulmonary carinoma. They may occur through either the lymph or the blood stream. The more frequent sites of metastases are the pleura, the liver, the regional and remote lymph nodes, other parts of the lungs, the kidneys, the bone, and the brain.

Symptoms of cancer of the lung are cough, sputum, hemoptysis, dyspnoea, cyanosis, dysphagia, pain in the chest, fever, loss of weight, weakness, cachexia, osteoarthritic changes in the extremities, particularly in the fingers, and sometimes chills, anorexia, nausea and vomiting. Hemoptysis is present in more than 50 per cent of the cases. It may vary in amount from slight streaking of the sputum to very severe hemorrhages. Dyspnoea also occurs in 50 per cent of the cases.

The physical signs are not characteristic. X-ray evidence is the most valuable single means of determining the presence and location of a pulmonary tumor.

The diagnosis of primary bronchial carinoma is very frequently not made during life. It is hoped that by placing emphasis on the occurrence of this disease, earlier diagnosis will be possible and perhaps lead to possible radical cure by surgery. Primary carcinoma of the lung must be differentiated from early tuberculosis, mitral stenosis, benign tumors of the bronchi, bronchiectasis, benign tumors of the lung, cysts or early pulmonary abscess by careful study of symptoms.

At the present little is known that is helpful in treatment. Preventive treatment seems the most promising method. Possibly better treatment of such chronic infections as pulmonary abscess, bronchiectasis and tuberculosis might prevent some cases from developing into carinoma.

—o—

DERMATOLOGY, X-RAY AND RADIUM THERAPY

Edited by C. P. Bondurant, M.D.
413 Medical Arts Building, Oklahoma City

The Problem of Pigment Formation. B. Bloch. Am. J. M. Sc. 177:609 (May) 1929.

The author gives the results of his experiments with the dopa reaction, which is the term he uses to designate the reaction of cells containing dopa-oxydase when placed in a 1 per cent solution of ¾ dioxyphenylalanine.

His conclusions are briefly stated as follows: In the higher vertebrates, in all places in which melanotic pigment, or melanoblasts are formed, the specific dopa reaction is found to be positive. The dopa reaction was positively related to the production of melanin, and the reaction was negative when this process was temporarily or definitely lacking. Dopa-oxydase, the cell agent responsible for the dopa reaction, was found to have the characteristics of an oxidizing ferment. The nature of the substances producing natural melanins is not know to the author exactly, but it is thought that pyrocatechin derivatives play a role in the natural pigment formation.

Sclerosing Treatment of Varicose Veins by Chemical Irritants. Longfeil and A. W. Dahlstrom, Am. J. M. Sc. 177:690 (May) 1929.

A sclerosing solution of invert sugar solution (invertose) was injected into varicose veins. The results were practically painless, free of sloughing in the case of extravenous injection and produced no general reactions.

The author records ninety-six cases of varicosities of the lower extremities, in which there were twenty ulcers, eight cases of eczema and eight with extensive edema. From 5 to 20 cc. of solution were used at each injection; the average number of injections was between five and six. In ninety-two cases, there were good results in two cases, fair results, and in two cases, the results were not known.

This method is believed to be the best, by the authors, because there is less danger of embolism, less cost, less pain, no scar, few recurrences and no loss of time to the patient.

The Bacterial Action of Roentgen Rays. Hirsh W. Sulkowitch, Bull. John Hopkins Hosp. 44:439 (June) 1929.

The authors have experimented with a small number of organisms with the results that different bacteria show varying degrees of resistance to x-rays. It is apparent that x-rays are deadly to bacteria. The procedures of the experiment with an adequate bibliography have been compiled upon the subject.

Allergic Purpura, H. L. Alexander and C. H. Eyermann, J. A. M. A. 92:2092 (June) 1929.

The authors report six cases of Hennoch's purpura. It was demonstrated that the condition of each case was caused by a sensitiveness to a particular foodstuff as milk, egg, potato, wheat, chicken, beans, lamb, red plums, onions, strawberries and eating-apples. No attempt was apparently made to desensitize the patients.

More Deaths from Thallium. Current Comments. J. A. M. A. 93:122 (July) 1929.

The deaths of three children from thallium poisoning is reported by Merkel in the Deutsche Zeitschrift dur die gesamte gerichtliche Medizin (13:237, 1929) and commented upon by the Journal. The three children, all below normal physically and mentally, were given the full amount of the drug in divided doses. It is pointed out that the drug should be given in one dose for ringworm of the scalp, and should be lessened for children who are below normal.

Incidence of Foot Ringworm Among College Students. Robert Legge, Lee Bonar and H. J. Templeton, J. A. M. A. 93:170 (July 20) 1929.

The first report on a survey of the incidence of ringworm was made at the University of California. It was found that 53% of tne men and 15% of the women were infected with ringworm. A year in college increased the per cent of infection to 79 among the men, and 17 among the women. This the second report made at the University of California. The women were provided a gymnasium with every known sanitary device while the men's gymnasium had none of the sanitary devices, nor were they so strictly supervised as the women. The women students were required to wear rubber bathing shoes and were not permitted to walk bare feet on the floors of the showers or runways to the swimming pools. This is thought to be the single important factor controlling the infection among the women.

Skin Lesions Among Tar Workers, H. B. Wood, J. Cancer Research 13:54 (March) 1929.

The author concludes that the production of tar papillomas and epitheliomas is due to chemical rather than physical irritation and that only the high temperature coal distillates contain carcinogenic substances. Anthracene oil is the principal high temperature content of coal tar. Low temperature distillate tars and petroleum tars are considered non-irritable. Pustular lesions are produced by the heavy oils yielded by the high temperatures in coke-making more readily than the low temperature oils but are not known to cause papillomas. The phenol oils cause dermatitis in susceptible skin. Among the handlers of coke tar at gas works, coke biproducts plants, briquette factories and tar paper and roofing paper factories, tar warts, without epitheliomatous changes were observed. There were a great number of tar warts, but no evidence of occupational cancer was observed. In gas works prolucing water gas and in briquette factories there was no evidence of tar warts.

ORTHOPAEDIC SURGERY

Edited by Earl D. McBride, M.D
717 North Robinson Street, Oklahoma City.

Etiology and Pathology of Chronic Deforming Arthritis. John V. Barrow and Eugene L. Armstrong. Calif. and West Med. XXVI, 323, Mar, 1927.

Although this paper was written by internists, it contains valuable points for orthopaedic surgeons because of the fact that the authors cooperate very intimately with the orthopaedic service in the Los Angeles General Hospital.

The deformity produced in this disease is in the nature of bone destruction, compensatory hypertrophy, and spastic irritation. The pathologic changes in the joint indicate that the etiologic factor proceeds from the endarterial tissue of the marrow toward the periphery. Clinical and therapeutic evidence indicates that the gasto-intestinal tract is the most plausible source of the toxins or organisms of lytic destructive powers which produce these joint changes. It is believed that the disease is a general systemic one and that while the joint symptoms are the most outstanding signs, there are many other parts of the body affected.

Of the 245 cases forming the basis of this paper, 235 were gastro-intestinal cases first. Of these, 71.6 per cent showed some abnormality of the bowels, such as constipation or diarrhoea. Protozoa were found in the intestinal tract in ninety-four per cent of the whole group of cases. In only two cases did the disease begin after acute tonsilitis; in thirteen cases after influenza and in the remainder, no history of acute infection of any kind could be obtained. Eighty patients had had all their teeth removed before coming under observation by the authors and in none had this procedure stopped the course of the disease. Tonsils had been removed in sixty per cent of the group with relief in only one case. Seventy-five patients had normal tonsils; eighteen, diseased. Only ten had bad teeth. In the course of the treatment, septic tonsils and teeth were removed but their removal made no striking difference in the progress of the disease.

The joints affected in the order of their frequency were finger and hands, knees, spine, ankles, hips, elbows, shoulders, wrists, sacro-iliac, feet and toes.

The treatment may be classified as (1) parastic, and (2) physiologic. The first consists in injections of emetin hydrochloride, 0.02, hypodermically every other day, every third dose intravenously over a period of three weeks, then twice a week for another three weeks, then weekly for a month or two. Salol-keratin coated ipecac pills are also given in many cases. Alcresta ipecac 0.4 in combination with calcium phosphate 0.25, is highly recommended, especially in cases with constipation. Dietary treatment failed to give results. The authors can not agree with Pemberton that glucose tolerance has anything to do with etiology or treatment.

Orthopaedic treatment is of no help if it makes no attempt to stop the cause of the pathologic condition.

In this groun, 209 cases were fairly treated and 30.6 per cent of these showed excellent results;

42.2 per cent good results; 20.5 per cent fair results.

A Clinical and Roentgenologic Study of High Colonic Irrigations as Used in the Therapy of Subacute and Chronic Arthritis. R. G. Snyder and S. Fineman, Am. J. Roentgenol, XVII, 27, Jan. 1927.

Emphasizing the frequent multiplicity of causes in the production of arthritis, Snyder believes that in refractory cases, after exhausting treatment of determined foci of infection, or failing to discover any foci, the poor therapeutic results are probably due to existence of infective foci or to toxic absorption in the genito-urinary and intestinal tracts. He feels that the genito-urinary tract has not received adequate attention. As to the colon, in spite of the absence of proof that stasis and infection here will produce chronic arthritis he has empirically used in the past four years, wherever clinically indicated, a system of treatment aiming at elimination and correction of intestinal stasis—this in addition to other accepted treatment. Since doing so, his clinical results have shown a definite improvement. He does not feel that the colon is a factor in all cases, no doubt in many cases it has no relation to the onset. By high irrigation is meant the insertion of a fifty-four inch or longer "Lockwood tube" passing into the colon as far as the splenic flexure or into the more proximal portions of it, as far as the caecum. Proper technique is of the greatest importance, and only by reference to the original article can this be understood. Only by x-ray can it be demonstrated that the tube has reached within the colon, and as this check was not feasible in all cases the authors cannot claim that all the benefit of the procedures in their cases was due to use of the high tube. A minimum course of twenty to thirty irrigations is insisted on; from two to four gallons each time; fresh cultures of B. acidophilus are injected into the colon after each irrigation.

"No implication is made that colonic irrigations are a specific cure for subacute and chronic arthritis. . . . . The improved clinical results, it would seem, should be attributed to the system of irrigations as a whole."

The article, of sixteen pages, includes a discussion which indicates that such procedure is work for the expert only.

Avulsion Fracture of the Os Calcis, Harold G. Lee. Boston Med. and Surg. J. CXCVI, 1000, June 6, 1927.

Alvulsion fracture of the os calcis, that is, the tearing away of the upper portion of the os calcis by action of the gastrocnemius-soleus muscles acting through the tendo Achillis, is a very rare lesion. Lee reports a case, with the treatment employed.

While leaning over, the patient, a heavy woman, lost her balance and fell. She suffered immediate pain in the left heel with inability to rise. Roentgenograms showed an avulsion fracture of the os calcis. Open reduction was advised and performed. At operation two lateral incisions exposed the broken fragment and the body of the os calcis. Chromic catgut sutures, No. 2, were passed through a drill-hole in the os calcis, and over the approximated fragment. These were tightly tied, holding the fracture in perfect position. The wounds were closed without drainage, and a plaster cast applied from toes to groin. X-rays taken five days after reduction showed perfect apposition. The cast was worn for three months, weight-bearing being allowed the latter portion of the time. At the end of six months bony union was complete, and the patient could walk up and down stairs without pain.

UNTOWARD EFFECTS OF TREATMENT BY PHENYLHYDRAZINE HYDROCHLORIDE

The untoward effects of treatment by phenylhydrazine hydrocloride were studied by Herbert Z. Giffin and H. Milton Conner, Rochester, Minn, (Journal A. M. A., May 4, 1929). In a series of forty-one cases in their experience since 1924, twenty-five patients have maintained a very satisfactory condition. Of the forty-one patients, eight have been able to control symptoms due to increased blood volume but are not in good health; in these, the disease is advanced and is accompanied by arteriosclerosis, and hepatic, cardiac and renal disease. Ten of the forty-one patients have died. Two of these presented a very favorable condition, but one met an accidental death and one died of pneumonia. The remaining eight deaths include the four reported here, and four others which apparently did not occur either during or shortly after the administration of phenylhydrazine. Four cases reported occurred in women more than 60 years of age, with advanced vascular and visceral changes. As a result of their experience with phenylhydrazine hydrochloride it would seem wise to declare the following principles of treatment: (1) Patients with advanced polycythemia vera of a grade necessitating confinement to bed should not receive phenylhydrazine. (2) Extreme caution should be observed in administering phenylhydrazine to patients more than 60 years of age, to patients who have marked arteriosclerosis, and to patients who manifest evidence of advanced visceral injury. It is wise to give a very small dose to such patients, possibly only 0.1 or 0.2 Gm., and to observe the effect over several days. This however, should not be done if the patients are bedridden. (3) Patients who have probably had thrombosis should be treated cautiously. (4) Every effort should be made to keep the already sluggish circulation as free as possible. Treatment is best carried out with the patient ambulatory. If a patient is under observation in a hospital, he should be kept on his feet as much as possible; massage and exercise in bed have proved to be satisfactory measures. (5) Excessive dosage in the initial course of treatment is not necessary; a total initial dosage of from 1.5 to 3.5 Gm., seems to be sufficient. The subsequent dosage can be determined by the patient himself in view of his symptoms, and patients with less advanced disease have done exceedingly well on from 0.1 to 0.3 Gm., of phenylhydrazine each week. Experience may indicate that the daily administration of the drug over a period of a week or ten days is not necessary, and that a small dose each week will be safer, and will be sufficient to control the symptoms and to maintain the efficiency of the individual.